



This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

- + *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.
- + *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.
- + *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.
- + *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at <http://books.google.com/>

LOCOMOTOR ATAXIA
WILLIAM J. M. A. MALONEY

LANE MEDICAL LIBRARY STANFORD
L409 .M25 1918
STOR
Locomotor ataxia (tabes dorsalis) : an i



24503339110

LANE



MEDICAL

LIBRARY

LEVI COOPER LANE FUND



1

LOCOMOTOR ATAXIA

(Tabes Dorsalis)

*AN INTRODUCTION TO THE STUDY AND
TREATMENT OF NERVOUS DISEASES,
FOR STUDENTS AND PRACTITIONERS*

BY

WILLIAM J. M. A. MALONEY, M.D. (EDIN.)

FELLOW OF THE ROYAL SOCIETY OF EDINBURGH; FELLOW OF THE NEW
YORK ACADEMY OF MEDICINE; FELLOW OF THE NEW YORK NEURO-
LOGICAL SOCIETY; NEUROLOGIST TO THE CENTRAL AND
NEUROLOGICAL HOSPITAL; FORMERLY PROFESSOR
OF NEUROLOGY, FORDHAM UNIVERSITY,
NEW YORK CITY



ILLUSTRATED

D. APPLETON AND COMPANY
NEW YORK **LONDON**

1918

EY

COPYRIGHT, 1918, BY
D. APPLETON AND COMPANY

LIBRARY

Printed in the United States of America

L409

M25

1918

TO
VICTOR EDGAR SORAPURE, M.B.,
F.R.C.S. (EDIN.),
SOMETIME
PROFESSOR OF CLINICAL MEDICINE
AND
PRO-DEAN OF THE FACULTY OF MEDICINE,
FORDHAM UNIVERSITY,
NEW YORK CITY

45327



PREFACE

As the brilliant researches of Sherrington upon reflex function, of Head, Holmes, Sherren and their co-workers upon sensory function, and of Langley, Gaskell, Biedl, and Gley upon vegetative function, have revolutionized our knowledge of nervous phenomena, it is now essential to restudy every nervous disease, to interpret its symptoms with the aid of this new knowledge, and to organize its treatment according to the new interpretation.

This book correlates our present anatomical, pathological, physiological, and psychological knowledge with reference to locomotor ataxia. The correlation has been planned solely with a view to treatment. Matters having no direct bearing on treatment do not, therefore, receive the attention which they usually enjoy in text books on nervous diseases.

As a student I suffered, in common with every other student, from inability to understand, through my training in anatomy and physiology, the clinical evidence of nervous diseases. So nervous diseases then interested me mainly as a compulsory exercise in memory, decreed by captious examiners. The student's lack of power to apply knowledge is recognized by all teachers. It is inherent to the phase of being taught. A mind attuned to acquire facts in bulk cannot simultaneously fix and correlate them.

After his preliminary training in the fundamental sciences of anatomy, physiology, pathology and psychology, the student should be taught, from a concrete example, how normal structure varies in nervous disease; how, from this anatomical variation, physiological and psychological consequences follow which make the symptoms inevitable and the treatment effective. With this training he will intelligently approach any nervous diseases he may subsequently encounter; he will become a thinking instead of an automatic physician, and nervous diseases will so interest him that much of their difficulty will disappear.

The student's royal road to the understanding of nervous diseases lies through tabes. In tabes there are demonstrable the

exciting cause—the spirochete, the lesions—the structural effect of that cause, and the physiological and psychological consequences of these lesions. Reflex integration and its disturbance, interruption of sensory and of motor paths, vegetative disorders, and the interaction of the psyche with the sensory, motor, and vegetative nervous systems, all can be presented connectedly and concretely to students through the study of tabes.

Indeed, there are few facts in neurology which cannot be demonstrated in tabes; and there are few nervous diseases so amenable to treatment as it is. The physiological and psychological basis of the treatment is obvious; the results seem often miraculous. Students trained in neurology through the teaching of tabes respect the science, value it, and practice it.

Hence I explain the physiology and the psychology of function in detail which would be unnecessary, were I writing solely for my present and not also for my future colleagues. I especially emphasize the rôle of the vegetative nervous system, partly because of our former neglect of it, but mainly because of the superlative importance it has not only in producing the symptoms but likewise in permitting the disease.

There is a psychological as well as a physiological element in all symptoms, in all disabilities. Psychological treatment is, therefore, as essential as physiological treatment. In many medical cases psychological treatment is the only treatment necessary, and in some of the others it is the only treatment possible. By those who ordain the medical curriculum, physicians are presumed to be endowed with the art of psychological treatment. We might as well be presumed to inherit the healing touch. Psychotherapy must be taught in the schools or medicine cease to be a legally restricted profession.

Upon the reciprocal relations of the mental state and the tabetic symptoms, and upon the interdependence of the symptoms, rests the basis of psychological treatment. Without knowledge of these relations, effective treatment is impossible.

Treatment based on these relations does not lead to radical cure, to the removal of lesions. But we can rarely remove nervous lesions; and often it is mainly the mental reaction to lesions which determines the pain or ease, the misery or comfort of patients. Psychic treatment counteracts unfavorable mental reactions and thus minimizes the influence of lesions. It further educates the patient to control residual symptoms,

to protect himself against their aggravation; and when such aggravation does occur it enables him quickly to alleviate it.

I systematize the application of this knowledge, for the principles upon which the tabetic's treatment is founded are not restricted in their usefulness to tabes. They are obviously applicable to the treatment of all conditions in which the mental state aggravates symptoms, or in which symptoms threaten the integrity of the mental state.

The method of treating ataxia which I have advocated since 1912, and which I now fully describe for the first time, is based on the thesis that perfect thinking is essential to perfect moving; and perfect moving is the outward sign of perfect thinking. Training in perfect thinking is, therefore, training in perfect moving; and training in perfect moving is training in perfect thinking. The details of the training and the measures needed to ensure optimal conditions for it, I have carefully outlined.

In this material and impious age the Jordan flows unheeded and the waters of Bethesda rise unwatched. Healing words replace healing waters. Religion and health are unhappily confounded by those who foster the Emanuel movement and by the earnest members of that faith which is disguised as science and called Christian. Where remorse is morbid, or suffering is due to ignorance of Divine love, ecclesiastical ministration is proper and pertinent; beyond these limitations, it is ultimately destructive to the faith which excuses it and to the health which it seeks. Medical inadequacy led to these movements; the measure of their success is the measure of our failure.

The cause of our failure is neglect of the study of the reciprocal relations of symptoms and mental states. The unauthorized, ignorant of the source of symptoms, but efficient in molding the mental state of the faithful, bring relief to those whom misguided medical effort has failed to ease. It is a matter of congratulation for all when suffering is relieved by any one. A sufferer has an inalienable right to seek relief at any source. We cannot continue to preëempt the power to heal when others succeed where we fail, nor can the community allow suffering to be subject to the ignorant.

To his science no less than to his conscience the physician owes the duty of giving mental ease to his patient. That ease must be given, not through stupefying with mystic incantations and irrational suggestions, but by rousing the patient to the na-

ture of his danger and by teaching him to organize his own defense.

Only through mental training will the sick unfailingly receive aid in defending themselves against the tyranny of symptoms. Defense based upon self-control over mental processes is more potent and permanent than that afforded by the degrading and treacherous support of suggestion, and more worthy and secure than that which is founded upon ignorant mysticism.

The source of each borrowed illustration is indicated in its caption. I am grateful to the several authors whose illustrations I have used.

I am indebted to Doctor Abrahamson, Professor of Clinical Neurology at Bellevue Medical School, Neurologist to the Central and Neurological, Montefiore, and Mount Sinai Hospitals, New York City. His great experience and knowledge were always at my service and were of invaluable assistance in many difficulties. Doctors Grossman and Wolf have earnestly worked with me and the former has kindly placed some excellent photographs at my disposal.

Years ago, when I was the Crichton Research Fellow, Doctor C. C. Easterbrook, the distinguished director of the Crichton Royal Institution, Dumfries, Scotland, stimulated me with his great enthusiasm for applied psychology. The length of my latent period is due mainly to the war; to that cause also, I hope, he, kindest of critics, will attribute what is lacking in the following pages.

Doctor H. R. Storer of Newport, Rhode Island, kindly corrected the proofs.

WILLIAM J. M. A. MALONEY

156 East 79th Street
New York City

CONTENTS

CHAPTER	PAGE
I.—THE SPIROCHETE AND TABES. THE SITE OF THE STRUCTURAL CHANGES PRODUCED BY THE SPIROCHETE IN THE CENTRAL NERVOUS SYSTEM	1

Discovery of tabes. Controversy concerning its cause. Relation of tabes to syphilis. The *Spirochaeta pallida*, the exciting organism of tabes. Strains of spirochete. Spirochetes that persist in the human host and attack tissues usually immune. The three varieties of the persisting spirochete. The attacking force of the persisting spirochete. Invasion by the persisting spirochete of vascular, glandular and nervous tissues. Causes which determine that the major attack shall fall upon the central nervous system. The spirochete's invasion not restricted to any one area in the central nervous system. Factors which determine the initial site, the extent and the direction of the spirochete invasion in the central nervous system.

II.—RESULTS OF THE SYPHILITIC LESIONS: DIS- TURBANCE OF THE REFLEX MOTOR FUNCTION	18
--	-----------

The posterior root ganglion and coverings. Ganglion cell lesions. Meningeal lesions. The posterior root itself a primary site of spirochete invasion. Nature and result of the syphilitic change in the posterior root. The sensory nerve of muscle. The simple reflex arc of muscle tone. The functional linking of reflex arcs. The knee-jerk, its relation to the reflex arc of muscle tone, its governing factors. Alterations of tendon reflexes in tabes. Hypotonia: effect on strength and mobility of joints. Dislocations. Contortions. Muscular weakness of tabes; causes. Disintegration of reflex function. Influence of extent, rapidity, site and duration of the lesions. Measurement of the amount of the disturbance. Identical motor effects produced by reflex and by cortical action. Site, paths and nature of cerebral control of movement. Reflex defects masked during voluntary movement.

CHAPTER

PAGE

III.—RESULTS OF THE SYPHILITIC LESIONS: DISTURBANCE OF SENSORY FUNCTION . . . 45

Course of the sensory impulses from the periphery to consciousness. Specific nature of the impulses conducted by sensory fibers. Sensory images. Factors which determine the fate of the sensory images. The peripheral impulse and the mental reaction to it. Measurement of stimuli. Sensory thresholds; their variability. Complex content of the sensation which arises from a simple stimulus. Sensory results of a complete and of an incomplete lesion of the posterior root. Selective invasion of the root fibers conducting pain. Touch loss. Temperature loss. Vibration loss. The postural sense. Paths of sensory fibers from the muscular apparatus and from the postural sense organ of the head. Loss of the sense of the body's posture. Measurement of the loss of postural sense. Effect of blindness and of training on postural sense in the non-tabetic and in the tabetic. Variability of postural sense loss. Postural sense loss inaccurate index of the severity of the tabetic lesions.

IV.—RESULTS OF THE SYPHILITIC LESIONS: DISTURBANCES OF ATTITUDE AND MOVEMENT . 61

The changing and maintaining of attitude. Attitude expressive of the motor tendencies that we desire and of those that are unconsciously evoked by the mental state. The impossibility of maintaining an immovable posture. Normal swaying. Tabetic swaying, Romberg's sign. The tabetic attitude. Effect of blindness, psychotherapy and mental states on the tabetic attitude. The tabetic attitude a consequence not only of the lesions but also of the mental state. Ataxic movements. Controversy regarding the cause of ataxia. Importance of the constituent parts of the muscular apparatus in coördination. Postural sense loss not parallel to ataxia. Reflex defect not necessarily associated with ataxia. Onset and course of ataxia. Fatigue, fear and shock as precipitants of ataxia. The ataxic moment. The relation of ataxia to the tabetic lesions. Psychic control of coördination. The ratio between effort and accomplishment in movement. Voluntary movement the resultant of the desired and of the unconsciously evoked postural images. The interaction of voluntary and of reflex movement. Psychic activity masking reflex defect in voluntary movement throughout the pre-ataxic stage. Ataxic complexes. Coefficient of ataxia. The

CONTENTS

xiii

CHAPTER

PAGE

reciprocal relations of ataxia and the mental state. The amount of disturbance of attitude and movement mainly a measure not of the structural changes but of the degree of the failure of conscious control of movement to compensate for the tabetic defect in the reflex mechanism. The surrender or so-called paralytic stage.

V.—RESULTS OF THE SYPHILITIC INVASION OF CRANIAL NERVES 86

The olfactory nerve. The optic nerve. Onset, course and nature of optic atrophy. Ophthalmoscopic findings. Psychic and structural factors in visual loss. Effect of mescaline in enhancing vision. Difficulty of measuring visual acuity. Measurement of light perception in nearly blind tabetics. Threshold of perception of light, threshold of perception of change. Blindness: frequency, time of onset, rate of development. Causes of spontaneous improvement in visual acuity. Absence of proof that optic tabes arrests spinal tabes. Influence of blindness upon ataxia. Spontaneous cure of ataxia following blindness. Influence of blindness upon the mental state and upon sensory perception. The relation of loss of vision to coördination. Effect of blindness upon pain and deafness. Third, fourth, and sixth nerves. Diplopia, its causes, squint, spontaneous cure. Means for ascertaining the presence of suppressed visual images. The fifth nerve. The seventh nerve. The eighth nerve. Vestibular lesions. Auditory mechanism. Psychic deafness. Tabetic deafness. Influence of deafness upon the mental state. Increase of deafness with stationary lesions. Other cranial nerves.

VI.—RESULTS OF THE SYPHILITIC LESIONS OF THE VEGETATIVE NERVOUS SYSTEM: SENSORY AND MOTOR DISTURBANCES 116

The correlation of cells. Development of the primitive vegetative nervous system. The vegetative nervous system in man composed of two antagonistic components, the vagal and the sympathetic. Action of these components illustrated by the pupil. The pupil in tabes. Vegetative reflex arcs and plexuses. The afferent and efferent fibers linking these arcs with so-called "centers" in the central nervous system. Tracing of the sensory fiber. Proof that pain from pressure on the face passes not by the fifth but by the sympathetic. Pres-

sure pain loss in tabes on limbs, eyes, testicles and other viscera. Loss of visceral reflexes. Lightning pains, characters and frequency: their source in irritation of the vegetative nervous system. Lightning pains and crises allied phenomena. Gastric, rectal, bladder and genital crises. Pains and crises reciprocally related to the mental state. The physiology of micturition. Micturition in tabes.

VII.—RESULTS OF THE SYPHILITIC LESIONS OF THE VEGETATIVE NERVOUS SYSTEM: DISTURBANCES OF CHEMICAL CORRELATION . . . 135

Action of the products of the internal secreting glands. The antagonistic reaction to chemical substances shown by the vagal and the sympathetic components of the vegetative nervous system. Oscillations in the balance between vagal and sympathetic components, due to chemical stimuli, control metabolism, and defense against organismal invasion. Disturbance of this control. Trophic conditions: ulcers, infections, fractures, arthropathies. Defense against the spirochete. Production of defense substances. Stimulation of vegetative nervous system by defense substances. Adequate stimulation causing perfect defense. Inadequate stimulation permitting persistence of the spirochete. Inadequate stimulation due to mildness of pretabetic lesions, to glandular insufficiency, to age, to mental stress. Imperfect defense, causes. Effect of lesions of vegetative nervous system upon defense. Why optic tabes is abortive. Why the vegetative nervous system, although the first, is not, as a rule, the main site of spirochete invasion.

VIII.—DIAGNOSIS AND COURSE OF TABES . . . 151

Symptoms and signs of tabes; their origin. Diagnosis of syphilis. First sign of invasion of nervous system. Course of invasion beginning in posterior root; beginning in optic nerve. Tabes diagnosed by evolution. Tabes a disease in which dominant symptoms arise from primary syphilitic invasion of posterior nerve roots. Pretabetic period, duration and significance. Effect of age on pretabetic period. Rate of syphilitic invasion. Rapid, slow and abortive invasions. Remissions. Preataxic stage. Ataxic stage. "Paralytic" or surrender stage. These stages psychological, not anatomical. Types of tabes. Age of onset of tabes. Termination of tabes.

CONTENTS

XV

CHAPTER

PAGE

IX.—ANTISYPHILITIC TREATMENT IN TABES . . . 165

Essential tabetic lesion irreparable. Tabes incurable. Effect of antisymphilitic treatment on the duration of the pretabetic period. Absence of proof that such treatment can prevent tabes. Nature of the evidence required to establish such preventive action. Absence of proof that antisymphilitic substances arrest tabes. Nature of the evidence required to establish the power of a drug to arrest tabes. Drugs commonly used in tabes. Mercury: modes of administration. Arsenic, enesol, salvarsan. Motives for intraspinal administration unfounded. The search for new preparations of antisymphilitic substances and for new methods of administration: indications of the inadequacy of these substances. Necessity for caution in the use of mercury and arsenic in tabes. Nucleinates and nucleinic acid.

X.—THE MENTAL STATE OF TABES . . . 176

Mental stress a cause of tabes. Tabes a source of mental stress. Tabes a cause of fear, of fatigue, and of depression. Psychoses in tabes. The psychosis typical of tabes an aggravation of the mental state induced by the tabetic symptoms. Resemblance between tabetic psychoses and prison psychoses. Measurement of the mental state of the tabetic. Weakening of attention in tabes: its sensory and motor effects. Variations in the mental state of tabes. Mental deterioration: its rate and its consequences. Influence of the tabetic symptoms upon the mental state. Influence of the mental state upon the tabetic symptoms. Estimation of the reciprocal relations of the tabetic symptoms and the mental state. Interdependence of tabetic symptoms. Foundations of mental treatment.

XI.—TREATMENT OF THE MENTAL STATE: TREATMENT OF THE SYPHILITIC NEURASTHENIC AND OF THE PRETAXIC TABETIC . . . 190

Imperative necessity for treatment of syphilitic neurasthenia, the precursor of tabes and paresis. Principles of treatment: rest, reduction of effort to capacity, avoidance of harmful thinking, training in mental control, freedom from unnecessary medical interference. Treatment of the pretaxic tabetic. Prosecution for syphilis. Communicating the diagnosis. Reducing mental expenditure. Isolation and

imprisonment. Immobilization and rest. Effect of attitude in perpetuating and aggravating stress. The teaching of rest. Rest exercises, respiratory and muscular. Psychological and physiological effect of the exercises, effect on blood pressure, pulse rate and pulse rhythm. Exercises in controlling mental processes through movement. Respiratory exercises, pneumograph. Head exercises, cephalograph. Eye exercises. Exercises in mental work. The reckoning test. The daily schedule. Results and duration of régime. The transition period. The return to business and social life. Systematizing and limiting daily effort. Importance of practicing the exercises to prevent relapses and to avoid ataxia.

XII.—PSYCHOTHERAPY OF PREATAXIC SYMPTOMS . 214

Preataxic symptoms. Diplopia. Pain, measurement and analysis. Treatment of subacute pain; of pain crises. Gastric crises, measurement, prevention. Treatment of pain crises. Cocain. Morphin. Root section. General principles of treating pain, gastric, and other crises. Involuntary micturition: prevention, treatment. Intestinal and genital symptoms. Blindness. Deafness.

XIII.—TREATMENT OF MOTOR SYMPTOMS OF TABES . 226

Faulty equilibration first motor disorder in tabes: its measurement, analysis, and prognosis. Ataxia, its prevention and measurement. Minimizing ataxia by reducing movement and by mechanical supports. Defects of the tabetic foot as a support. Means of ascertaining and of remedying these defects. New method of obtaining cast of foot in action. Foot brace. Special tabetic shoe. Braces for joints. Value of belt and bandages. Temporary use of all appliances.

XIV.—TREATMENT OF DISORDERS OF MOVEMENT IN TABES 244

Removal of muscular constraint. Easy, confident, and perfect movement founded upon appreciation of postural images. Measures to insure appreciation of postural images. Blindfolding. Training in attention and in capacity for effort. The maintenance of optimal conditions throughout the treatment. Training in attitude, recumbent, sitting, "all fours," kneeling, standing. Dosage of postural exer-

CONTENTS

xvii

CHAPTER

PAGE

cises. Training in movement in these several attitudes as they become stabilized. The cardinal qualities of simple movements. Simple movements. Complex movements. Stabilizing the base for progression. Progressing. Creeping. Kneeling. Walking. Dosage of movement exercises. Walking in public.

XV.—REVIEW OF TABES 264

Afferent nervous system invaded in disease styled tabes dorsalis, or locomotor ataxia. Disturbance of vegetative, non-vegetative and sensory function. "Locomotor ataxia" misleading; "tabes dorsalis" useless; "syphilitic neuritis of posterior roots" suggested. Diagnosis and course of invasion. Division into preataxic, ataxic, and paralytic stages a psychological, not anatomical, division. Drug treatment of tabes. Reciprocal relations of symptoms and mental state in tabes and interdependence of symptoms. Treatment of the symptoms through the mental state. Its ease and effectiveness. Treatment of the mental state through the symptoms. Former methods, Frenkel, Goldscheider, Förster. Limitations of Frenkel's method. Development of present method. Value of present method in cure and in prevention. Conclusion.

BIBLIOGRAPHY 277

INDEX 287

LIST OF ILLUSTRATIONS

FIG.	PAGE
1.—Spirochetes in the brain of a general paralytic	3
2.—The Spirochaeta pallida in the brain of a general paralytic	4
3.—Spirochaeta pallida, from the brain of a general paralytic, growing in the testes of a rabbit	6
4.—Beaussart's quoted series	8
5.—Beaussart's recorded series	9
6.—Diagram to show the relations of the spinal cord and its coverings to the anterior and posterior nerve roots and to the peripheral nerve	19
7.—Muscle spindle and motor end plate, diagrammatic	21
8.—Tendon organ, diagrammatic	21
9.—Reflex collaterals of posterior root	22
10.—Diagram of a simple reflex arc	23
11.—Diagram of the nerve mechanism which correlates the action of antagonistic muscles	25
12.—Diagram to show a subminimal impulse in one reflex arc reinforced by a subminimal impulse in another reflex arc	26
13.—To show relations of the sensory nerves of the musculature to the cerebellum	29
14.—Graphic record of the knee jerk to show its true relations	31
15.—Demonstrating early hypotonia of the flexor muscles of the knee of a recumbent tabetic	33
16.—Left genu recurvatum, showing the importance of muscular action in the production of this deformity	34
17.—Excessive tabetic hypotonia	35
18, 19.—Backward dislocation of right knee	36
20, 21.—Syphilitic invasion of anterior horn cells in a tabetic	38
22.—Chimpanzee's brain	40
23.—Diagram showing the course, origin and termination of spinal cord fibers	41
24.—The three stages in the normal grasp	43
25.—The tabetic grasp showing asynergia	44
26.—Anatomical arrangement of the paths and centers concerned in sensation	45
27.—Von Frey's hairs	47
28.—Table of von Frey's hairs	48
29.—Algesimeter	49
30.—Pressure esthesiometer for testing pressure touch	50

FIG.	PAGE
31.—Modified Cattell algometer for measuring pressure pain	51
32.—Muscle spindle	54
33.—Tendon organ	54
34.—Ruffini's corpuscle, from ligament of knee	55
35.—Goldscheider's instrument for measuring joint movements	57
36.—"The mood, the emotion, the mental state, at the moment— have motor attributes of which we are unconscious"	63
37.—Tracing of lateral swaying in a normal person	63
38.—Tripod stage of a tabetic	64
39.—Quadruped stage of a tabetic	64
40.—Further development of the "quadruped" stage	65
41.—"The courageous can always maintain, although distortedly, an erect posture"	66
42.—Dr. Abrahamson's case	99
43.—Paralysis of the left third nerve	105
44.—Complete paralysis of the fourth nerve and partial paralysis of the third	106
45.—Complete paralysis of the right sixth nerve	108
46.—Complete right and partial left ptosis in tabes	109
47.—Diagrams to show the effect of locomotor ataxia on the pupil	117
48.—Retraction of the lids, eccentric pupils	118
49.—Tabetic with left pupil widely, right moderately dilated	118
50.—Extirpation of Gasserian ganglion	120
51.—Extirpation of Gasserian ganglion	121
52.—Extirpation of Gasserian ganglion	123
53.—Left facial palsy from a basal fracture	123
54.—Right facial palsy of acute inflammatory origin	123
55.—Diagram showing relation of sympathetic fibers	124
56.—Diagram of autonomic or vagal and sympathetic interaction	136
57.—Trophic ulcer of the big toe	137
58.—Trophic ulcer of the outer margin of the foot	138
59.—Taboparesis	139
60.—Painless tabetic fracture of the head of the tibia	140
61.—Charcot left knee and ankle	140
62.—Tabetic joints	141
63.—Charcot elbow	142
64, 65.—Proliferating osteitis of lower left ribs in a tabetic	143
66.—Experimental brain syphilis in a rabbit	144
67.—The reckoning test	179
68.—The reckoning test of A. R.	180
69.—The reckoning test of J. H.	181
70.—Description of parts of pneumograph	196
71.—Parts of recording apparatus of pneumograph	197
72.—Types of breathing disturbance	198

LIST OF ILLUSTRATIONS

xxi

FIG.	PAGE
73.—Types of breathing disturbance	199
74.—Types of breathing disturbance	200
75.—Types of breathing disturbance, Curve A	200
76.—Types of breathing disturbance, Curve A 2	201
77.—Types of breathing disturbance, Curve A 3	202
78.—Types of breathing disturbance, Curve A 4	204
79.—Types of breathing disturbance, Curve A 5	205
80.—Types of breathing disturbance, Curve A 6	206
81.—Types of breathing disturbance, Curve A 7	206
82.—Chart of patient beginning treatment, showing by the reek- oning test, remarkable improvement in her capacity for simple mental tasks	208, 209
83.—Chart of gait in early case of locomotor ataxia	228
84.—Footprints in the slightly ataxic	230
85.—Showing initial state of a tabetic	231
86.—Broad base and crouching attitude of tabetic	232
87.—Footprints of tabetic	233
88.—Footprints of tabetic	234
89.—Footprints of tabetic	235
90.—The tabetic's boot	238
91.—The tabetic's boot	239
92.—Showing bandages used as temporary museles	240
93.—Showing bandages used as temporary museles	241
94.—To show effect of treatment	241
95.—To show effect of treatment	242
96.—Knee guards	257
97.—To show effect of treatment	274

LOCOMOTOR ATAXIA

CHAPTER I

THE SPIROCHETE AND TABES. THE SITE OF THE STRUCTURAL CHANGES PRODUCED BY THE SPIROCHETE IN THE CENTRAL NERVOUS SYSTEM

Discovery of tabes. Controversy concerning its cause. Relation of tabes to syphilis. The *Spirochaeta pallida*, the exciting organism of tabes. Strains of spirochete. Spirochetes that persist in the human host and attack tissues usually immune. The three varieties of the persisting spirochete. The attacking force of the persisting spirochete. Invasion by the persisting spirochete of vascular, glandular and nervous tissues. Causes which determine that the major attack shall fall upon the central nervous system. The spirochete's invasion not restricted to any one area in the central nervous system. Factors which determine the initial site, the extent and the direction of the spirochete invasion in the central nervous system.

AFTER hazy recognition by Todd (1847) in England and by Romberg in Germany (1841-51), tabes was discovered by Duchenne of Boulogne and its symptoms definitely separated from those of other diseases in his classical work on "*Ataxie locomotrice progressive*" (1857-58).

Since Duchenne's day, Charcot, Déjerine, Egger, Fournier, Pierre Marie, Marinesco, Nageotte and Raymond in France; Erb, Förster, Friedrich, Goldscheider, Leyden, Moebius, Oppenheim, Strümpell and Westphal in Germany; Argyll-Robertson, Byrom Bramwell, Graham-Brown, Buzzard, Ferrier, Gowers, Hughlings Jackson and James Taylor in Britain; Jelliffe, Mills, Noguchi,

Patrick, Spiller and Spitzka in America; Krafft-Ebbing and Schaffer in Austria; and Frenkel in Switzerland, have by their genius and labor conferred upon us most of our present knowledge of tabes.

The cause of tabes has been the subject of speculation and controversy almost from the discovery of the disease. Erb and Moebius stoutly upheld the syphilitic origin of tabes. Fournier believed not only that syphilis is the cause of tabes but also that he could gauge from the nature of the banal lesions of syphilis the probability of the later occurrence of tabes. Hitzig taught that tabes is due to a toxin liberated somewhere as the result of accident. Others incriminated shock, overwork, heredity and diverse causes.

With Schaudinn's discovery of the *Spirochaeta pallida*, the protozoön that causes syphilis, renewed interest in this controversy arose. Failure to find the spirochete in the tabetic lesion and lack of success of drug treatment directed against the spirochete in tabes led most clinicians to agree that tabes was due to a toxin. This toxin was believed by the majority to be a by-product of the spirochete's activity; by Brissaud to be liberated from the lymphocytes. Tabes free from all taint of syphilis was still thought to occur, and tabes not causally but accidentally associated with syphilis was supposed to be possible.

Wassermann proved that a substance detectable in the blood and in the cerebrospinal fluid may result from infection by the *Spirochaeta pallida*. Its presence practically signifies syphilis but its absence does not preclude syphilis. McIntosh and Fildes found the Wassermann substance in the cerebrospinal fluid of over ninety per cent, and Hauptmann and Nonne in one hundred per

cent, of cases of tabes. Spirochete infection is, therefore, an invariable accompaniment of tabes. There is no tabes without syphilis.

Vedder ascertained by blood examination that seven-

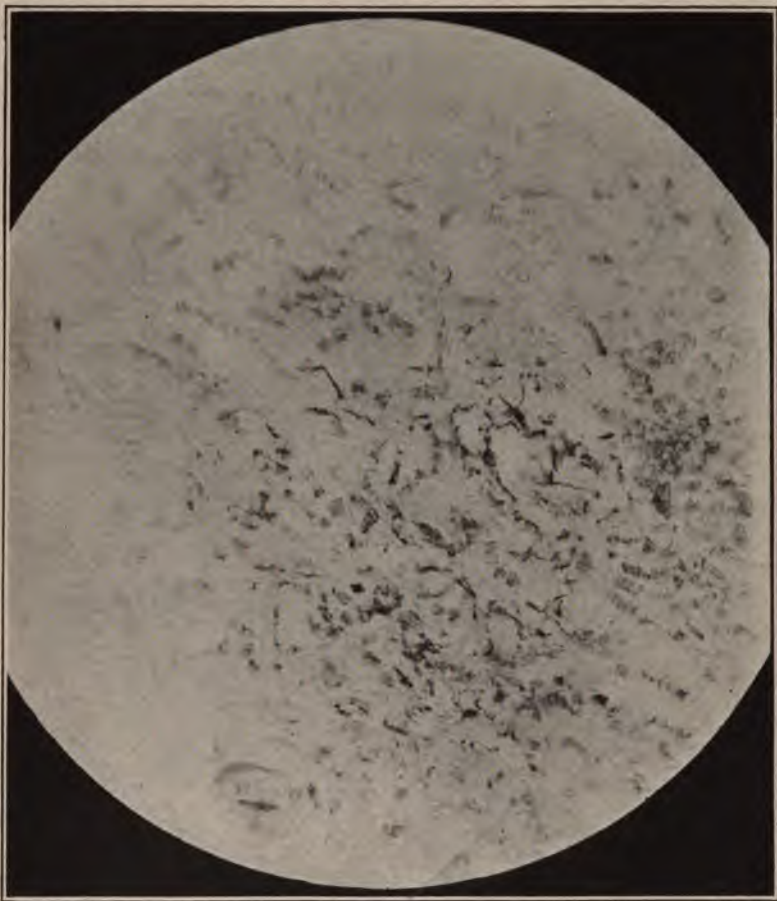


FIG. 1.—Spirochetes in the brain of a general paralytic. (Noguchi.)

teen per cent of the recruits of the United States Army are syphilitic. As syphilis occurs only in seventeen per cent of the non-tabetic and in one hundred per cent of the tabetic, syphilis has a causal relation to tabes.

In tabes a degeneration of the nerve processes and a

proliferation of the cells of the blood vessels and of the stroma occur. These lesions are of a nature compatible with syphilitic origin.

Pathologists now recognize, at the starting point of

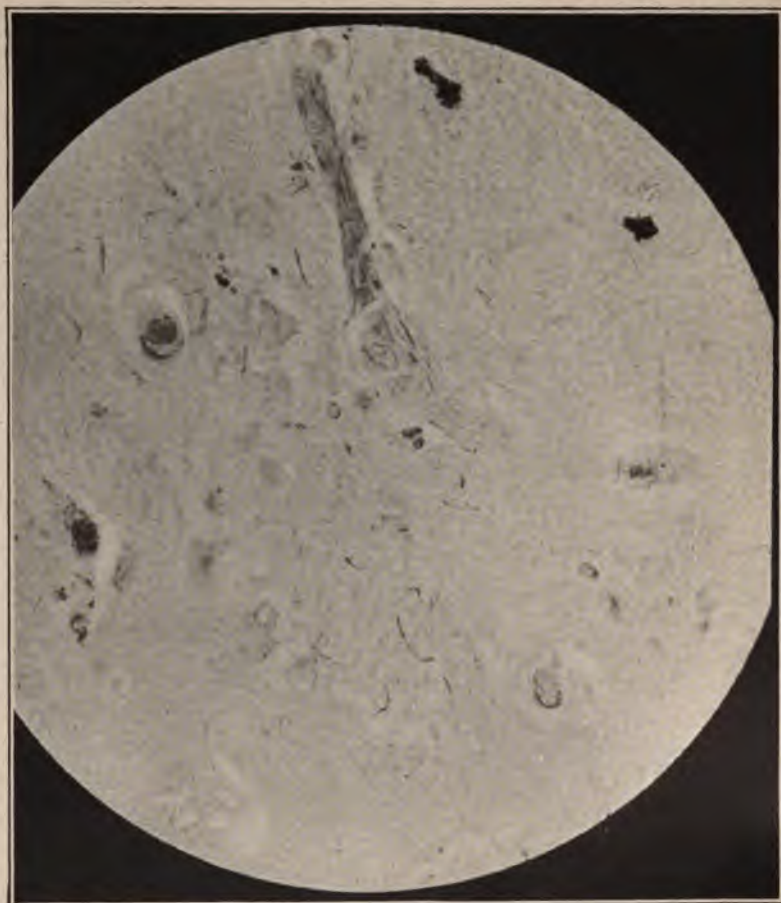


FIG. 2.—The *Spirochaeta pallida* in the brain of a general paralytic. $\times 1100$. (Noguchi.)

every tabetic change, an exudative focus. Exudates are evidence of inflammatory, not of toxic, action.

In the tabetic exudate Noguchi and others found the spirochete viable and culturable.

No organism other than the spirochete has been found in the inflammatory foci of tabes.

As syphilis is invariably present in tabes, as syphilis has a causal relation to tabes, as the lesions of tabes are such as could be produced by the spirochete, as the spirochete has been detected in the tabetic lesions, and as it is the only known organism which has this constant association with tabes, the *Spirochaeta pallida* must be the exciting organism of tabes.

The Wassermann substance can be detected in the blood a few weeks after the primary inoculation: according to Wile and Stokes, Nichols and others, the majority of the cases of syphilis in the secondary stage and many in the primary stage show evidence of the infection in the cerebrospinal fluid also. The local inoculation with syphilis, therefore, early becomes a general infection.

When the infection is general the tissues are equally accessible. The tissue first attacked must, therefore, be that which affords the most favorable soil for the spirochete's growth. The skin and the mucous membranes are the contact surfaces of human beings. From the skin and mucous membrane lesions of one host originate lesions of the skin and mucous membranes of another host. The most frequent and often the only sign of the family taint in the wife and child of a tabetic or of a general paretic is a lesion of the nervous mechanism of the pupil; from a spirochete growing in the nervous system of one host, a spirochete growing only in the nervous system of another host may result. The spirochete in a new host grows best in the tissue from which it is derived. The strain of spirochete is somewhat specific for the tissue it invades, for the tissue in which it grows. McDonagh and Ross state that these strains may be morphologically

differentiated; except variations in viability and in the rate of growth in cultural media, no definitely distinctive characteristics have been found by other observers, including Noguchi, Nichols, and Reasoner, to separate these strains.

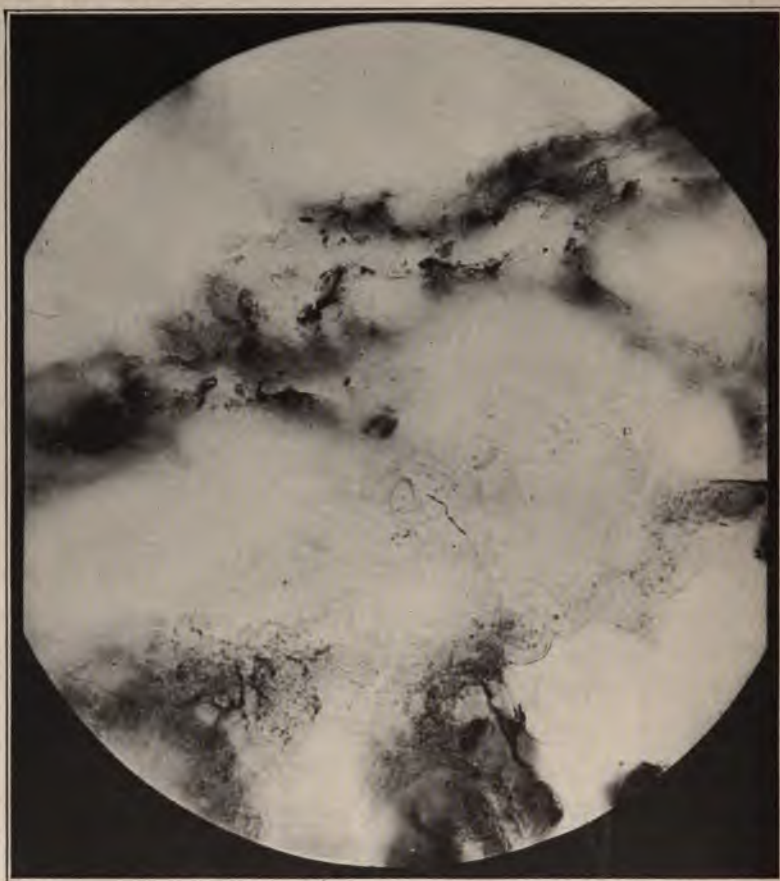


FIG. 3.—*Spirochaeta pallida* from the brain of a general paralytic growing in the testis of a rabbit. (Noguchi.)

When a person acquires a spirochete from contact with skin or mucous membrane lesions, the infection soon becomes general and shortly thereafter lesions appear in that person's skin and mucous membranes, heal and re-

cur; but till the last has finally healed, none as a rule is seen in the deeper tissues.

When the emasculated and less numerous spirochetes invade the deeper tissues, lesions occur there but not elsewhere: the gumma develops alone, unaccompanied by either skin or mucous membrane lesions or by lesions of the central nervous system. So well recognized is this incidence that the rare lesions which contravene the rule are styled precocious. The central nervous system lesions do not occur together with lesions of the deep tissues, nor of the skin and mucous membranes. Spillman and Perrin have reported the occasional occurrence of gummata and even of cutaneous syphilids with tabes and with general paresis; but such rare happenings serve merely to emphasize the rule that only one type of tissue is attacked at a time.

Few syphilitics enter matrimony ere the skin and mucous membrane lesions heal. The healthy partner is infected by a spirochete already repelled from the skin and mucous membranes of the infecting partner, a spirochete which can grow with difficulty or not at all in the skin and mucous membranes of the new host. The mildness of the skin and mucous membrane lesions in conjugal syphilis is notorious. The spirochete of the gumma and of the tabetic lesion so rarely produce skin and mucous membrane lesions in another host, that tertiary and nervous lesions are commonly considered to be non-infective.

The spirochete to which a particular tissue or tissues in one host have achieved immunity loses the power to attack that tissue or tissues, not only in the host where the successful defense occurred, but also in any other host, probably to the extent to which the biochemical re-

actions of the same tissues in the two hosts are identical.

The spirochete acquires in the human host characters which enable it to grow best in a certain tissue. If that tissue be the skin and mucous membranes, the spirochete, after growth there, may grow in the deeper tissues; and, still later, in the central nervous system. If that tissue be not the skin and mucous membranes, then the spirochete's growth is impossible there, but is possible in the deeper tissues, and later in the central nervous system. If that tissue be the central nervous system the spirochete may grow best and only there.

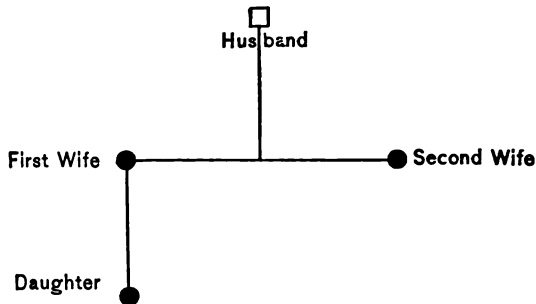


FIG. 4.—Beaussart's quoted series. □, Non-pervous syphilis; ●, syphilis of the central nervous system.

One strain of spirochete which may produce tabes is a spirochete which can grow best or only in the central nervous system; a spirochete which is derived from a host in whom it has been successfully repelled from the skin and mucous membranes and deeper tissues; a spirochete which causes tabes after no or slight pretabetic lesions, a spirochete which is partly blamable for the many cases of tabes that are ignorant of their infection, and for the long accepted but erroneous belief that tabes can develop without syphilitic infection.

Belières records three cases of general paresis and two of an allied nature that all owed their infection to

the same woman. Beaussart quotes a series in which a syphilitic husband, free from nervous affection, had a wife who became tabetic, and a daughter who was a general paretic. After divorce, the husband remarried and his second wife also developed tabes.

Beaussart records a series in which a glassblower, X., acquired a buccal chancre from a drinking cup used by a fellow workman, Y. X. and his wife died of general

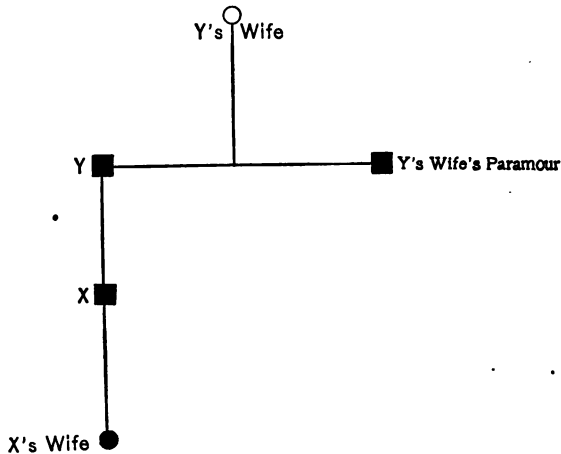


FIG. 5.—Beaussart's recorded series. ○, Non-nervous syphilis; ■, ●, syphilis of the nervous system.

paresis. Y. died of general paresis. Y.'s wife remained normal but her paramour developed tabes.

Beaussart mentions a man who became a general paretic and whose wife showed taboparesis: her lover developed general paresis and his wife had syphilitic pupillary changes. Morel Lavallée records five cases of general paresis, and Erb five cases of tabes, with a common source of infection. Pierre Marie and Bernard and others have reported similar groups.

In such groups no single source of infection, far less an unique exposure to syphilis, can be established. Yet

the larger the group or series of similarly affected cases the greater is the probability that their common experience is also the source of their common mischance. From a common source organisms of an uniform virulence are derived. If in spite of possible differences in dose, in treatment, and in defense, a series of persons infected from a common source acquire nervous symptoms, either a spirochete with a particular faculty to invade nerve tissue is the infecting agent, or several persons, reacting to the ordinary spirochete in the same extraordinary way, are, by a remarkable coincidence, infected from the same source.

Belières' report makes no mention of nervous affection in the woman alleged to have transmitted the taint; in Morel Lavallée's series the alleged source of infection did not herself suffer from syphilis of the nervous system; in Beaussart's recorded series, Y.'s wife had no implication of the nervous system. To establish the existence of a special strain of spirochete which can triumph over the average human resistance and finally attack the nervous system, it is, however, not necessary for all exposed to that spirochete to develop syphilitic nervous lesions. If in one series of syphilitics the proportion of cases of nervous disease is greater than the usual proportion in which nervous disease follows syphilis, then a special strain may be acting in that series.

A second analogous series weakens the possibility that persons reacting to spirochete infection in the same unusual manner were coincidentally infected, at the same source, by an ordinary spirochete, and increases the probability that the reaction which characterized the series is due to a special invasive power of the spirochete infecting them in common. Every additional series

immeasurably enhances this probability. Even the few I have cited demonstrate that there are one or more strains of the spirochete which characteristically persist in spite of the average human resistance, which attack the nervous system, and which maintain this power of attack in transmission from host to host. Perhaps the person in any group free from nervous lesions is the original source of the group infection.

The persisting spirochete is one which can overpower the average human resistance; or one which, being of average virulence, encounters subnormal resistance; or one which evades the usual resistance by its inability to attack the outpost tissues.

The persisting spirochete has an attacking force which depends upon its numbers and virulence. With this force the persisting spirochete attacks tissues which are usually safeguarded. Among these protected tissues the central nervous system does not suffer alone; the cardiovascular and glandular systems are also invaded. The cardiovascular system is invariably attacked: the attack is usually ignored because of the more bizarre and insistent results of the concomitant nervous lesions. But many authors have emphasized the frequency of cardiovascular, especially aortic, lesions in tabes; Matsunaga found fifteen cases of cardiac disease among ninety-nine tabetics. Burr, Grossman, and others have shown the importance of cardiovascular and renal lesions in bringing death to the tabetic. Strümpell has demonstrated the frequency with which patients suffering from aortic and other vascular lesions of syphilitic origin show signs, such as fixed pupils and loss of tendon reflexes, of concomitant syphilitic affection of the central nervous system.

Strains of spirochete which grow best in the central nervous system; poisons, such as alcohol and tobacco, which lower the vitality of the nerve elements; and constitutional, environmental and occupational influences which render the nervous the most vulnerable system, are the factors that decide that the major attack of the persisting spirochete shall fall on the nervous system.

$$\text{Liability to syphilis of} \quad \text{Attacking force of} \\ \text{central nervous system} = \frac{\text{spirochete}}{\text{Inherited} \pm \text{acquired} \\ \text{resistance.}}$$

In the central nervous system the spirochete has no exclusive site of attack.

At least ten per cent of tabetics show optic atrophy. Galezowski found posterior root lesions in 717 out of 1029 optic atrophies. Mott states that fifty per cent of cases of syphilitic optic atrophy end in general paresis. Tabes may change to general paresis. Déjerine states that more than half the cases of general paresis show signs of implication of the posterior roots.

The syphilitic invasion called tabes may advance till much of the central nervous system is implicated.

Oppenheim found in all of the fourteen cases of tabes which he examined endoneuritis or perineuritis in the peripheral sensory nerves. There may be one or several foci in a nerve. Such foci may occur in the secondary stage of syphilis and may not be followed by tabes. Each focus may implicate few or many fibers. The resulting destruction is usually partial and often insignificant. Nonne and others have examined cases in which they failed to find lesions in the peripheral nerves.

Jendrassik, who had a cerebral theory of ataxia to substantiate, declared that a special area of the cerebral

cortex was characteristically implicated in tabes. Lesions all over the cortex have been since reported. General paresis may early or late supervene in tabes. Gordon Holmes has shown that tabes may occur without any cortical lesions.

The oculomotor and abducens nerves are usually affected, less often the trochlear; the vagus much more frequently than the fifth; but all the cranial nerves have been found implicated both in their course and in their nuclei of origin.

Together with the vestibular branches of the eighth, and independently, the cerebellum may be attacked.

Not only the vagal but also the sympathetic component of the vegetative nervous system may be extensively diseased in tabes. Of the sympathetic fibers those in the posterior roots are usually attacked. The dorsal sympathetic, as a rule, suffers first and most.

Tabetic foci have been seen in the anterior horn cells as well as among their homologues in the basal nuclei. Such foci are rare and occur late. S. A. K. Wilson states that they are accidental complications of tabes. Lesions in motor nerves have also been occasionally observed.

Homen and Marinesco demonstrated the presence of the spirochete in the lymphatic channels of peripheral nerves. Orr and Rows showed that a communication exists between the general and the cerebrospinal lymphatic systems, at the posterior nerve roots; and that a continuous upward flow of lymph occurs along the peripheral nerves and through the posterior roots. Hence the invasion of the posterior roots was attributed to their accessibility to the spirochete. Poirier found a similar communication in the optic nerve sheath which seemed to explain the liability of the optic nerve to invasion.

But in the olfactory sheath, also, there exists such a communication: another is present in the eighth nerve sheath; and still others exist where tabetic lesions are infrequent. As the spirochete invasion early becomes general; as sites which enjoy with the posterior roots and optic nerves a communication between the general and the cerebrospinal lymphatic systems do not share with these an equal liability to attack by the spirochete; and as, moreover, Guillain and others, by injection of coloring matter, have demonstrated a constant circulation occurring in the cerebrospinal lymphatic system, the distribution of the spirochete probably plays an insignificant rôle in determining the site of the initial attack.

The main stress of ergot poisoning falls on the posterior roots; of amyl alcohol on the optic nerve; of strychnin and of tetanus toxin, probably, on the synapse of the anterior horn cell. Only local differences in the chemical composition of the nervous system can account for the selection by these several poisons of their respective sites. Such chemical differences may render cultural conditions at one site favorable, at another site less favorable, to the growth of organisms.

Some cells, owing to developmental characteristics, may be less resistant than others; cells whose processes are unprotected by a neurilemmal sheath may be more vulnerable than others which are sheathed.

Edinger fatigued rats by placing them on a continuously moving treadmill. He then poisoned them with pyridin and found the lesions, although disseminated, mainly in the posterior root zones. In Professor Kraepelin's laboratory, one eye of certain rodents was closed. The open eye was then exhausted by bright flashes of light, recurring every minute, for several

weeks. After injecting amyl alcohol into these rodents, one found the optic nerve of the open eye showed greater destruction than that of the closed. It has, therefore, been proved that nerve elements exhausted by excess of functional activity resist less than unfatigued nerve cells, the action of toxic agents. We may infer that neurons exhausted by over-function resist less than those not so fatigued, invasion by organisms.

The amount of stress to which a neuron is subjected depends upon its function and upon its owner's environment. The functioning of sensory neurons, particularly of those which constitute the posterior roots, is almost incessant; whereas, that of motor neurons is intermittent. Sensory neurons are commonly, motor rarely, attacked by the spirochete.

Only unconvincing evidence has been yet offered to prove the influence of occupation in determining the site of the initial lesion, for neurologists have, as a rule, confounded disability due to psychic, with that due to structural, causes. But Hitzig, Oppenheim, and others have emphasized that among the posterior roots those of an injured limb are first attacked; and the frequency with which the vagal and the sympathetic components of the vegetative nervous system are over-stimulated from the mental stress which conduces to tabes may explain why these components are so often found degenerated in tabes.

Where the spirochete finds the most favorable soil in which to grow and the lowest resistance, it commences the invasion.

$$\text{Liability of a neuron to invasion} = \frac{\text{Attacking force of spirochete.}}{\text{Defensive power of neuron.}}$$

When an optic nerve is attacked, that optic nerve is at the moment of invasion less capable of defense than the

other optic nerve, than the spinal roots, than the cerebral cortex and than any other site.

When two sites in the same nerve, or in different nerves; two optic nerves; two spinal roots; an optic nerve and a spinal root, are equally vulnerable they are simultaneously attacked.

When the attack shifts, it shifts to the next most vulnerable site. It is noteworthy that when the disease starts in one optic nerve, the next site attacked is usually the other optic nerve. Similarly, when the posterior roots are first attacked the disease tends to spread among posterior roots in preference to other sites.

If the optic nerve be the first attacked, the lesions there are usually more severe than if the optic follow spinal lesions.

Perhaps the spirochetes acquire a predilection for sites chemically identical with those wherein they first successfully grow in the nervous system. But sometimes the attack halts in, say, the spinal roots, and begins in the optic nerve; the resistance of the optic nerve then has fallen lower than that of the spinal roots.

In the central nervous system, as in other tissues, the lesions of syphilis tend to appear in crops separated by periods of outwardly perfect quiescence.

After a completely, or incompletely, successful attack on the posterior roots or on the optic nerves, no further lesions may occur. Then the spirochete either has perished or has been rendered finally quiescent: competent seekers have so far failed to find the spirochete in the optic lesions. If no other spirochetes exist active in that host, the spinal roots are spared attack. In this sense, and only in this sense, does optic tabes arrest spinal tabes, does spinal tabes arrest optic tabes. The pres-

ence or absence of lesions in the optic nerve or at any other site in no way renders other sites more fragile or more resistant. Optic atrophy has no more influence in arresting spinal tabes than has general paresis.

With one exception, which we shall later (page 50) consider, the site first attacked is usually the chief site of the disease, but the longer the invasion lasts the less the primary tends to be the sole site.

Much labor has been expended in determining the frequency with which various sites are exclusively and in their combinations attacked. Such frequency may vary widely. Thus, Déjerine states that muscular atrophy occurs in 20 per cent of cases of tabes: Duchenne, in 8 per cent and Eulenberg, in 0.6 per cent. Each of these percentages is doubtless correct, but only for the particular group of cases examined by its observer. Déjerine's group comprised many cases of long standing; Eulenberg's comprised many recent; hence their statistics differ.

There is no spirochete which can attack only the posterior root, or only the cortex and not other sites in the central nervous system. By the symptoms which are dominant we clinically identify the syphilitic invasion; and the disease conserves its identity just so long as this dominance is maintained.

CHAPTER II

RESULTS OF THE SYPHILITIC LESIONS

1. DISTURBANCE OF THE REFLEX MOTOR FUNCTION

The posterior root ganglion and coverings. Ganglion cell lesions. Meningeal lesions. The posterior root itself a primary site of spirochete invasion. Nature and result of the syphilitic change in the posterior root. The sensory nerve of muscle. The simple reflex arc of muscle tone. The functional linking of reflex arcs. The knee-jerk; its relation to the reflex arc of muscle tone; its governing factors. Alterations of tendon reflexes in tabes. Hypotonia: effect on strength and mobility of joints. Dislocations. Contortions. Muscular weakness of tabes; causes. Disintegration of reflex function. Influence of extent, rapidity, site and duration of the lesion. Measurement of the amount of the disturbance. Identical motor effects produced by reflex and by cortical action. Site, paths and nature of cerebral control of movement. Reflex defects masked during voluntary movement.

THE spinal cord is built of segments each of which possesses an anterior and a posterior root. On every posterior root is a mass of nerve cells, called a spinal ganglion. Every cell of a spinal ganglion is bipolar; that is, has two processes, a peripheral and a central. The peripheral processes constitute the common sensory nerves, and in skin, muscle, tendon, cartilage and other tissues terminate as specialized sense organs, called receptors.

The central processes, passing to the spinal cord, constitute the greater part of the posterior nerve root. The posterior nerve root is covered by a prolongation of the meningeal covering of the spinal cord.

In tabes the bipolar cells of the spinal ganglion have

been seen in all stages of disintegration. The cell changes may be primary or may be secondary to lesions in their peripheral or in their central processes. A lesion of its peripheral process produces more profound changes in a bipolar cell than a lesion of its central process. Changes in the bipolar cells are seldom extensive and may indeed be undetectable (Schaffer).

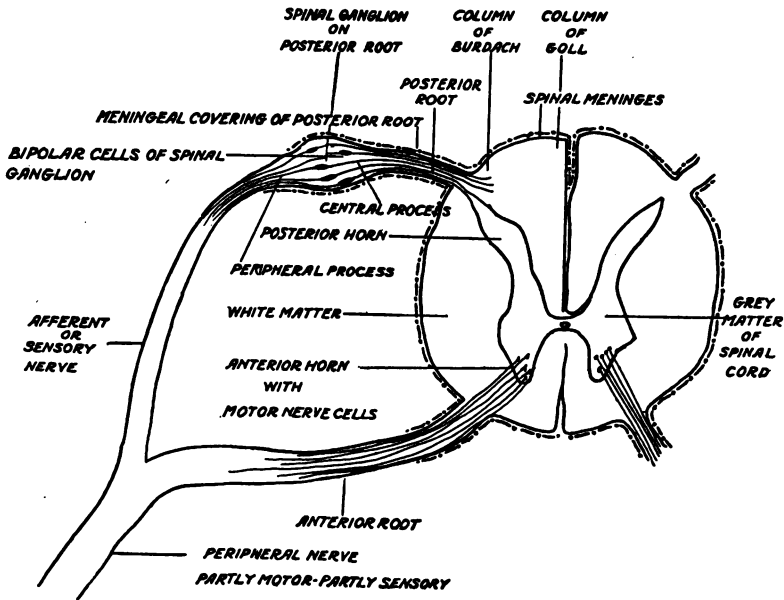


FIG. 6.—Diagram to show the relations of the spinal cord and its coverings to the anterior and posterior nerve roots and to the peripheral nerve.

The meningeal coverings of the posterior roots may be opaque and thickened and the meninges elsewhere normal. More commonly, the thickening spreads to the spinal cord where scattered patches of chronic meningitis may also occur, especially in advanced cases. But the meninges may be unaffected.

Pierre Marie attributed the degeneration of the posterior root fibers in tabes to the destruction of the bi-

polar cells from which they originate in the spinal ganglion. But as the earliest degeneration in the posterior root is more extensive remote from, than near to, the spinal ganglion, the degeneration of the posterior root fibers is not a consequence of the destruction of the ganglion cells. Indeed, degeneration of the posterior roots not rarely is unaccompanied by any spinal ganglion lesions.

Others have stated that degeneration of the posterior root fibers is a result of an inflammation of the root's coverings. The site of the greatest degeneration is usually not in the fibers immediately under the meninges. Severe meningitis may be associated with slight degeneration of the root fibers; severe degeneration may be associated with slight meningitis or even with no meningitis. Hence posterior root degeneration may occur independently of meningitis.

The posterior root lesion may occur in absence of lesions in the spinal ganglion and in the meninges. The posterior root is, therefore, a primary site of syphilitic invasion. This initial posterior root lesion may be augmented from foci appearing synchronously or subsequently in the spinal ganglion or in the meninges. The meninges and the spinal ganglion cells may be affected independently of, or secondarily to, the posterior root.

The reaction in the posterior root to the spirochete invasion is not solely a degeneration of nerve fibers. Here, as elsewhere in the body, no organismal attack on the essential cells can occur without a defensive response in their supporting tissue. In the stroma which supports the nerve fibers, the lymphocytes and mast cells congregate around the attacked focus, the fixed tissue cells multiply, and the endothelium proliferates in the

vascular and lymphatic channels. Indeed, Nageotte, from careful studies, concludes that the change in the posterior root is a transverse interstitial neuritis. Fur-



FIG. 7.—Muscle spindle (left) and motor end plate (right), diagrammatic. (Regaud, Graham Brown.)

ther histological examination with the Nissl methods perfected by Alzheimer are needed to confirm or disprove Nageotte's conclusion; but such examinations in the optic nerve in tabes have shown the change to affect primarily the nerve processes and secondarily the interstitial tissue (Stargardt).

Beginning probably among the middle zone fibers (Schaffer), the syphilitic invasion spreads to affect other fibers of the posterior root. Roots may be attacked simultaneously or in succession. The disease is said to begin usually in the lumbosacral roots, sometimes unilaterally, as a rule bilaterally. It spreads early to the dorsal, later to the cervical, roots.



FIG. 8.—Tendon organ, diagrammatic. (Regaud, Graham Brown.)

In these regions it advances, most destruction occurring, commonly, in the lumbodorsal region. One or several or the majority of the posterior roots in a region may be implicated. A root may be wholly destroyed; generally some of its fibers escape. A fiber may have one or more diseased foci.

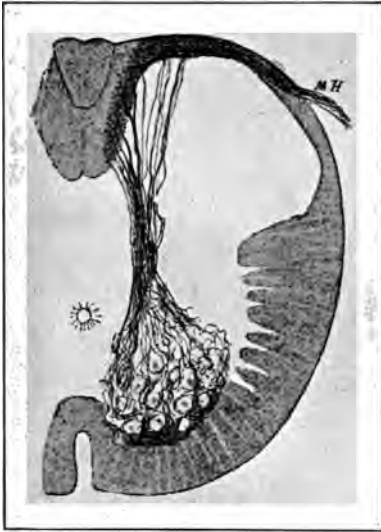


FIG. 9.—Reflex collaterals of posterior root (Lenhossek, Graham Brown.)

Every fiber of the posterior nerve root, which is a central process of a bipolar cell of a spinal ganglion, when it reaches the cord, divides into two main branches, an ascending and a descending; and these give off many lateral twigs called collaterals.

A syphilitic lesion in the posterior root may sever this central process. On the peripheral side of such a lesion a fragment

of the central process is left attached to the ganglion cell: but the greater part, lying on the spinal side of the lesion, is, with all its branches, then cut off from its cell of origin, its center of nutrition in the spinal ganglion; and, in consequence, degenerates. Degeneration occurs simultaneously throughout the severed portion, stem, branches and twigs.

The sensory nerve of voluntary muscle originates, as the other sensory nerves do, in a bipolar cell (a^2 , Fig. 10), in a spinal ganglion, on a posterior nerve root. The peripheral (a^1 , Fig. 10) of the two fibers of this cell ends

within a muscle as a fusiform structure which is called a muscle spindle (a, Fig. 10). The central fiber passes along the posterior nerve root, to the posterior zone of the spinal cord, where it gives off a lateral branch, the reflex collateral (a⁵, Fig. 10). This reflex collateral traverses the gray matter of the cord, from the posterior horn to the anterior, to form a junction called a synapse, with a nerve cell there (a⁶, Fig. 10).

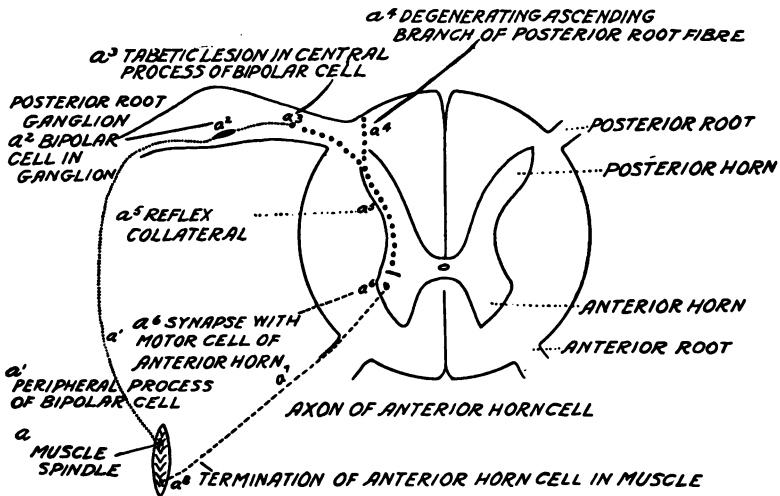


FIG. 10.—Diagram of a simple reflex arc. Tension changes arising in the muscle, affect the muscle spindle where they are converted into nerve energy which normally passes in impulses from the muscle spindle along the sensory nerve a¹, a², a³, a⁵ to a⁶, and is there transformed into motor nerve energy which passes back to the muscle by a⁷ to the motor end plate a⁸ at which physico-chemical changes are initiated in the muscle. The effect of a tabetic lesion at a³ is depicted

This junction is an intimate contact only; at it, continuity of nerve substance is not established; it is an articulation, not a fusion; the surface of contact at the synapse is also the surface of separation. The physiological degeneration of the severed spinal portion of the posterior root fiber extends to the termination of every

branch of that fiber, but no further. The degeneration reaches the synapse and stops there.

The cell with whose processes the synapse is formed does not degenerate. Changes, however, do occur in it. These were formerly thought to be tabetic but have now been shown to arise from disuse and to occur also in experimental cutting of the posterior root.

The axon (a^7 , Fig. 10) of this anterior horn cell leaves the cord by the anterior nerve root to terminate (a^8 , Fig. 10) within the muscle in which the muscle spindle lies.

The muscle spindle (Fig. 7) is so formed that it is specially sensitive to the tension changes which occur within a muscle during stretching, contraction and relaxation. Acting as a receptor of these changes, the muscle spindle transmutes them into nerve energy which passes in impulses along the afferent peripheral nerve, through the posterior root, and the posterior horn of the spinal cord, to the synapse with the anterior horn cell. A special afferent path (a , a^1 , a^2 , a^3 , a^5 , Fig. 10) is thus constituted for the impulses evoked by tension changes in muscle. These impulses are not merely transmitted by this path but they are also converted in it from sensory to motor impulses. This conversion occurs at the synapse. The anterior horn cell with its axon forms the efferent path (a^6 , a^7 , a^8 , Fig. 10) along which the motor impulses are discharged to induce changes in the muscle.

An afferent sensory path and an efferent motor path linked in this way constitute a reflex arc. In this reflex arc non-sentient stimuli from the muscle are converted into physicochemical action, which purges the reflex arc of them. Such physicochemical action induces in the muscle increase, or diminution, of tone or irritability.

When a muscle is stretched, its tone increases; when relaxed, diminishes. Upon the tone of the muscle its readiness and its power to contract, and also its fatiguability, depend.

This reflex arc, although ideally composed of a single

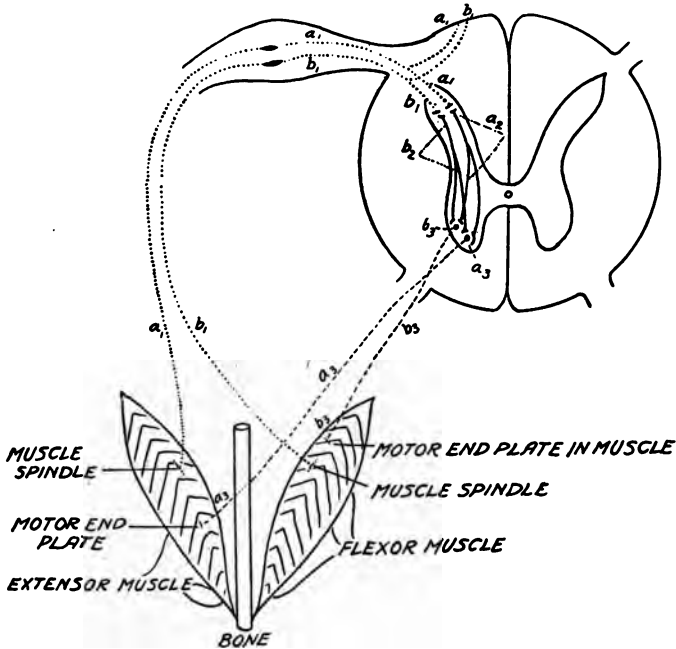


FIG. 11.—Diagram of the nerve mechanism which correlates the action of antagonistic muscles. Reflex arcs, *a* and *b*, extensor and flexor arcs respectively, depicted, each with one alternative path, to show reciprocal innervation of anterior horn cells a_3 and b_3 .

afferent limb, is not really so simply built. Between the reflex collateral and the anterior horn cell one (a^2 , Fig. 11) or more synapses may be intercalated; the afferent limb, instead of being a single fiber, then consists of several fibers (a^1 , a^2 , Fig. 11), along which impulses from the muscle are relayed. Moreover, a synapse may be not a mere relay station on an inevitable route, but a junction common to several routes (a^3 common to a^2 and b^2 , Fig.

11) each of which is a possible path for impulses brought by the rest. Some of the remaining collaterals of the posterior root fiber are linked ultimately with anterior horn cells, other than that with which the reflex collateral joins.

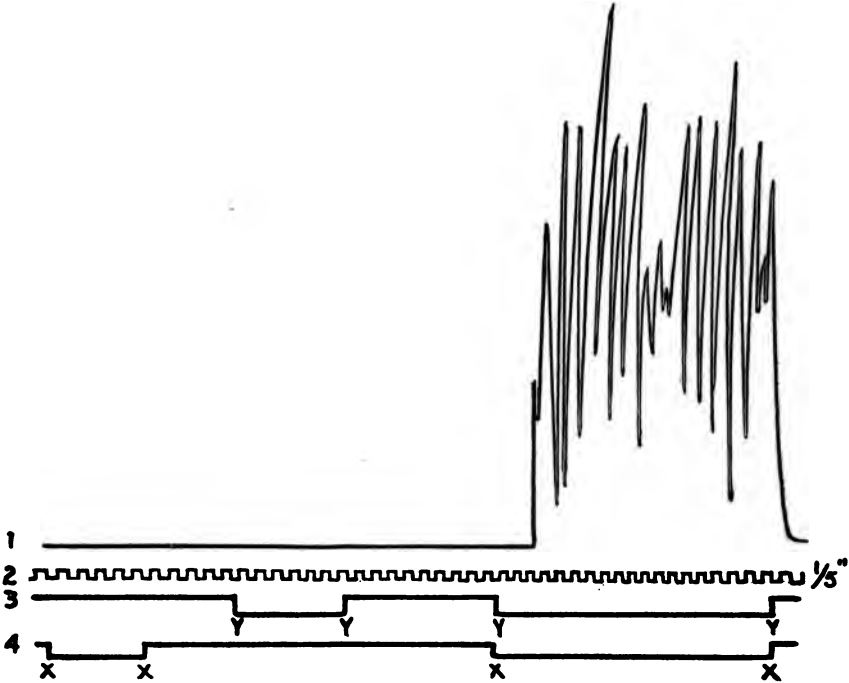


FIG. 12.—Diagram to show a subminimal impulse in one reflex arc reinforced by a subminimal impulse in another reflex arc, so that both together produce a response which neither singly can elicit. 1, line of muscle curve; 2, time marked in fifths of a second; 3, line indicating the excitation of the one reflex arc; 4, line indicating the excitation of the other. A subminimal stimulus $x-x$ was first given to the one; no muscular response followed. Next, a subminimal stimulus $y-y$ was given to the other; no response followed. Then $x-x$ was applied to the one while $y-y$ was simultaneously applied to the other; the result was the rhythmic muscular contractions depicted. (Modified slightly from Sherrington.)

Therefore, every primary arc has associated with it alternative paths for its impulses; and every motor nerve cell may be excited by impulses other than those proper to the particular reflex arc of which it forms the efferent

limb: there is reciprocal innervation of the motor nerve cells in the cord; entering sensory impulses radiate through the cord.

This radiation is not haphazard. The resistance encountered by the impulses entering the cord determines the direction in which they radiate. An impulse flows along the path of least resistance; and the amount of flow in alternative paths is inversely proportional to the resistance these paths offer. The seat of the resistance is the synapse. In a moment of inaction among the reflexes, the path of least resistance to an entering impulse is the primary reflex arc of the impulse.

A stimulus, unless it has a definite minimal intensity, elicits no reflex response; but repeated stimuli of an intensity less than this minimal are summed in the arc until a response is produced. Subminimal impulses serve to lower the resistance of the synapse, to facilitate the passage of succeeding impulses.

After a motor response, the reflex arc is for a time markedly less excitable. During this refractory period, the synapse resistance is so high that the further passage of impulses is blocked. The afferent impulses are then diverted from their usual reflex path to the alternative paths which momentarily offer less resistance than the primary reflex arc. These alternative paths lead to other motor nerve cells (to b^3 instead of a^3 and vice versa, Fig. 11), one of which is adequately excited and gives a motor response. In that reflex arc a refractory period ensues; and the direction of flow is again changed. In addition to the alternate facilitation and inhibition of the passage of stimuli in the reflex arc, motor response is further correlated by its constant and rhythmic nature.

When the extensors of the knee contract, the flexors simultaneously relax. According to McDougall, during the excitation of the extensor reflex arc, a (Fig. 11), the resistance at the synapse, a^2 , a^3 , is lowered to such an extent that impulses traveling in the flexor arc, b , impulses upon which the tone of the flexor depend, finding their usual path, b^1 , b^2 , b^3 , offers higher resistance than the alternative path, b^1 , b^2 , a^3 , take the latter path. The flexor impulses are drained from the flexor arc and the tone of the flexor muscle falls. As flexor action antagonizes extensor, the relaxation, or loss of tone, in the flexor muscles permits extensor movement with a minimum expenditure of energy: inhibition of the reflexes which subserve flexor tone normally synchronizes with facilitation of the extensor reflexes.

In bending the elbow, when the arm hangs at the side, muscular action first fixes the shoulder, and then, while the forearm makes with the upper arm a smaller and smaller angle, moves the upper arm backwards. This harmonious movement of the bony levers of the skeleton is called a synergic movement. Among the reflexes implicated in such a movement, activity in one lowers the threshold of the next; and each succeeding reflex in the chain has its threshold lowered in sequence, so that the motor responses follow one another in regular order and the associated reflexes constitute a functional unit.

Resident in the reflex mechanism of the spinal cord is thus a vast integrating power of which the unit is the reflex arc and which coördinates the muscular activity of the individual and of the allied segments of the body.

The ascending branches of the sensory fibers of the muscles, in their course through the posterior columns of the cord, are accompanied by the fibers from every

receptor of the tendons, bones and joints. These segregated, ascending branches of the fibers of the muscular apparatus give off collaterals which form synapses with cells of the columns of Clarke (a^1 , Fig. 13) from which

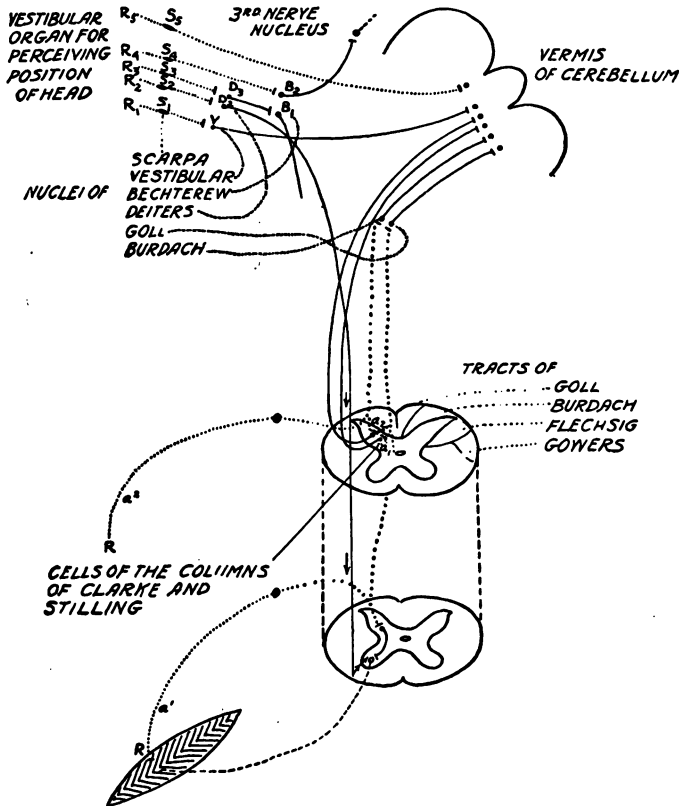


FIG. 13.—To show relations of the sensory nerves of the musculature to the cerebellum. R = receptor. For explanation see text.

arise fibers that pass in the tract of Flechsig (Fig. 13) to the cerebellum; and also other collaterals which form synapses with cells of the column of Stilling (a^2 , Fig. 13) from which arise fibers that pass in the tract of Gowers, likewise to the cerebellum. The ascending branches themselves all end by forming synapses among the cells of

the nuclei of Goll (Fig. 13) and of Burdach (Fig. 13) in the medulla, whence some axons pass, mainly uncrossed, to the middle lobe of the cerebellum. Non-sentient impulses from the muscular apparatus pass, therefore, by two paths to the cerebellum, via a collateral and a spino-cerebellar fiber, and via the ascending branch and a medullary-cerebellar fiber.

In each internal meatus lie the bipolar cells of the ganglion of Scarpa. Their peripheral processes form in the labyrinth a sense organ which is attuned to receive variations in the pressure of the otoliths and of minute masses of fluid in the utricle, saccule and semicircular canals. The variations are initiated by change in the position of the head. The central processes constitute an afferent nerve, the vestibular nerve.

The non-sentient of the impulses from the labyrinth sense organ pass along the vestibular nerve, through Deiters' nucleus (Fig. 13, $R_2S_2D_2$) and through the nucleus of Bechterew (Fig. 13, $R_4S_4B_2$) to the anterior horn cells of the spinal cord and to their homologues among the cranial nuclei. These impulses may thus be distributed to every reflex arc of muscle tone. The labyrinth reflexes adjust the position of the eyes and of the head to the horizon. The attitude of the head reflexly determines that of the other parts of the body.

The vestibular organ of tone is connected both directly (Fig. 13, R_5S_5), and through the cells of its subsidiary nuclei (Fig. 13, $R_1S_1V_1$), most intimately with the cerebellum.

The cerebellum receives impulses from every receptor of the muscular apparatus in the limbs, trunk and head. It sends out impulses to every motor cell. The cerebellum is the central organ of the non-sentient of the impulses

which arise in the muscular apparatus; it is the center of integration not only of the segmental reflexes but of the reflexes of the whole musculature: it is thus the center of muscular tone; of dynamic action; of synergic action; of equilibration; and of every other form of reflexly correlated muscular action. By virtue of cerebellar control not only do the parts of a limb act harmoniously in movement but the limbs move in orderly

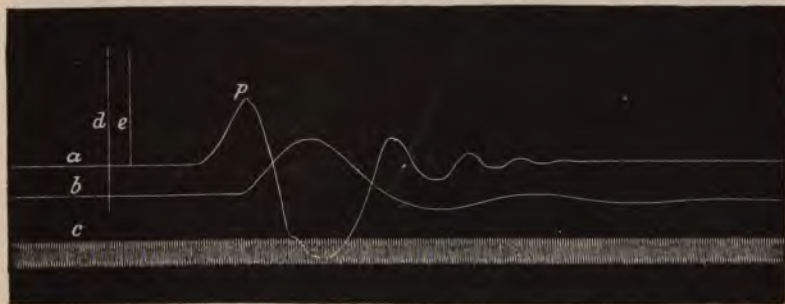


FIG. 14.—Graphic record of the knee jerk to show its time relations. (Weiler.) *a*, records the movements of the hammer used to tap the patellar tendon in eliciting the knee jerk; *p*, marks the moment the tap was administered; *b*, traces the curve of the leg movements; *c*, is the time record in $\frac{1}{100}$ seconds. Note the initial positions of the writing points did not coincide; *d*, indicates the initial position of the writing point of the leg movement, *e*, that of the writing point of the hammer movement.

relation to one another, and to the trunk; the body in motion maintains its equilibrium; and the entire musculature is a functional unit.

The tone of a muscle depends not merely on its local reflex arc but also upon the segmental reflexes allied with it, and upon the vestibular reflex: all are under the control of the cerebellum, which is subject to cerebral regulation.

The tone of a stretched muscle is raised, of a relaxed muscle is lowered. If the tone of the quadriceps be increased and that of the flexors diminished by bending

the knee, a tap on the patellar tendon will cause a quadriceps contraction which will extend the leg. This phenomenon is called the knee-jerk. The extent of the jerk varies somewhat with the tap but mainly with the tone of the extensors and the relaxation of the flexors. Formerly we believed that the interval between the tap and the jerk was shorter than the time necessary for reflex action and that the jerk was due to a direct response of the muscle. Weiler showed conclusively that the interval was ample (0.050 to 0.070 second) for reflex action and that the knee-jerk was indubitably a reflex act.

As no contraction occurs unless the tone of the quadriceps be adequate, and as muscle tone is maintained by reflex activity, the knee-jerk affords an index to the integrity of the reflex arc of tone of the quadriceps.

The briskness, the fatiguability and, in part, the extent of the knee-jerk depend upon the quadriceps tone. The tone of the quadriceps is less in fatigue than in freshness, after repose than after moderate exercise.

The activity of the cerebral cortex diminishes muscular tone. Indeed, hemiplegia, supervening on tabes, may cause the reappearance of a lost knee-jerk. The less attention is directed to the knee, the more does the tone of the muscles there correspond to the local conditions of stretching and relaxation. Measures to distract the patient's attention, to reinforce the knee-jerk, are many. Among them, those most used are engaging him in conversation; directing his gaze upwards; making him, at the word of command, squeeze the physician's arm; and inducing him to hook the flexed fingers of his up-turned hand on those of his down-turned hand, and then to hold the one fixed and to pull strenuously with the other.

In the exhaustion of cortical activity due to the mental

stress which usually precedes tabes, cerebral control of reflexes diminishes. The pretabetic shows, therefore, abnormally active tendon reflexes. Tendon reflexes that respond equally on both sides, apart from absence, or almost absence, may indicate no structural abnormality. But a unilateral change in the extent, the readiness, the briskness, or the fatiguability is always pathological.

The posterior root lesion early and almost invariably



FIG. 15.—Demonstrating early hypotonia of the flexor muscles of the knee of a recumbent tabetic.

implicates the afferent fiber of the reflex arc of muscle tone. The particular arc affected depends upon the root diseased. Thus lesions of the first and second root affect the ankle-jerk, the so-called Achilles reflex; of the third and fourth lumbar roots, the patellar reflex or knee-jerk; of the cervical roots, the tone reflexes of the arm muscles; of the motor root of the fifth, the tone reflex of the jaw. The Achilles reflex is usually first affected, then the knee-jerk. The arm-jerks may be involved later in the disease. Usually they escape altogether; rarely they are the first affected. Only the reflex on one side may at first be attacked. As early cases are

not often seen, bilateral implication is most common. The various tendons reflexes may be affected in all possible combinations.



FIG. 16.—Left genu recurvatum, a case showing the importance of muscular action in the production of this deformity. The patient had a shortened right lower limb owing to an old fracture of the femur which united in a bad position. Hence, she mainly used her left leg in walking.

When the afferent of the reflex arc of muscle tone is attacked, irritation precedes destruction. Exaggeration, therefore, precedes loss of the tendon reflexes. This transient exaggeration is not often seen. Destruction quickly follows irritation and the jerk becomes of smaller and smaller extent until it can be elicited only by reinforcement; then it finally disappears.

The hypotonic muscles tend less and less to resist stretching: they lengthen. In joints whose main support is muscular, abnormal mobility, deformity and weakness result. Thus, at the wrist, hypotonia permits unusual degrees of flexion and extension. An abnormal backward bending of the knee in standing develops

(*genu recurvatum*, Fig. 16). In the foot the antero-posterior arch soon yields. Frequently the tabetic can assume attitudes as contorted as those seen in markedly rachitic children (Fig. 17). The weakening of joints

sometimes leads to dislocations, especially backward dislocation of the knee (Figs. 18 and 19). The elongated muscle must first in contracting shorten to its normal length before it can begin to act on the bony levers. This



FIG. 17.—Excessive tabetic hypotonia permitting a contorted attitude similar to that sometimes assumed by rachitic infants.

mechanical disadvantage is minimized by the speed of muscular contraction.

The antagonists do not adequately and synchronously relax when the agonists contract. In the thigh of a recumbent, muscular tabetic who is endeavoring to extend the flexed leg, one may sometimes feel the struggle between the contracting extensors and flexors: to the rigidity of both are added shifting areas of contraction, according to the dominance of which the leg halts



Fig. 18



Fig. 19

FIGS. 18 AND 19.—Backward dislocation of right knee (Fig. 18); x-ray photograph of knee of same (Fig. 19).

or jerks onward. The strength of the movement is that of the excess of extensor over flexor action. The effort to move is increased, its effectivity is diminished.

The anterior horn cell is the trophic center of the muscle. This cell may undergo disuse changes owing to the destruction of the reflex collateral. It may be directly attacked (S. A. K. Wilson). This direct attack is rare and usually late. When only advanced cases of tabes were identified, Déjerine stated that twenty per cent of tabetics incur muscular atrophy. Eulenburg, in five hundred cases at all stages, found this formidable percentage reduced to 0.6. The usual march of change in electrical excitability is evident in the muscles in these cases.

Besides the anterior horn cell, its axon in the anterior nerve root or in the motor nerve may be implicated. To such lesions Déjerine attributes the tabetic "*pie d bot.*" Others have described a persistent contraction of the muscles of the palm (Figs. 20 and 21).

But besides these factors, there is in the muscular weakness of tabes a factor upon which stress has not been laid, a factor of paramount importance, a mental factor. This mental factor shows itself either in the readiness with which fatigue arises, or in the trifling muscular power called forth by voluntary effort compared with that apparent in automatic movement. Many cases have been described in which muscular fatigue or weakness was an early, a prominent, but commonly a transient symptom. Sometimes, in the tabetic, an absence of power similar to that seen in hysteria is evident (*see page 182*). By suitable therapy, strength may usually be quickly restored.

When the syphilitic lesion in the posterior root com-

pletely severs the central process of the bipolar nerve cell, the degeneration stops at the first synapse, the cell



Fig. 20

of which undergoes no consequent change except that caused by disuse. Subsequent relay fibers of the various



Fig. 21

FIGS. 20 AND 21.—Syphilitic invasion of anterior horn cells in a tabetic causing selective wasting among the thumb muscles, a flattening of the thenar and hypothenar eminences and a “clawing” mainly of the ulnar fingers. (Jelliffe.)

reflexes within and between the segments, connecting and commisural, spinocerebellar and medullary-cerebellar fibers, do not degenerate. Destruction of any one of

a series of relay fibers is physiologically equivalent to a lesion of the succeeding relays, in so far as the function of the succeeding relays depends upon that of the destroyed. Thus, a syphilitic lesion in the posterior root causing destruction of the collaterals which arise from the ascending branch and pass to the columns of Clarke and Stilling (a_1 , a_2 , Fig. 13), is physiologically equivalent to a lesion of the tracts of Flechsig and of Gowers, which is equivalent to a lesion of the cerebellum.

The lesion in the posterior root usually severs some fibers completely and others incompletely. To the extent of the severance, reciprocal innervation of the anterior horn cells is abolished. Fewer, feebler and delayed impulses pass the lesion to reach the branches or collaterals, to attain the connecting fibers; and the radiation of impulses is lessened. Among the reflex arcs associated with the affected fibers, radiation facilitates the passage of impulses. With the lessening of radiation, facilitation diminishes and its regular sequence in the affected arcs fails. In the synchronous or successive exciting of allied muscles and in the inhibiting of their opponents, disorder thence arises. A loss of strength, of rhythm, and of harmony now becomes evident in muscular action.

The disintegration of reflex function varies according to the rapidity of the nerve destruction. The more rapid the destruction the more marked is its disturbing effect. But the first is not necessarily the permanent effect. When a number of paths are linked in a common function the sudden destruction of one path may temporarily disorganize the whole function. But the remaining paths may quickly adapt themselves to the loss of their fellow and to a considerable degree compensate for it.

The disturbance of reflex integration produced by lesions differing only in site, is most severe when the lesion is in the cerebellum; more grave when in the vestibular organ than when in the spinal cord; and when in the spinal cord, is less and less important the further the affected roots are from the head segment.

Every movement is a part of a greater movement,

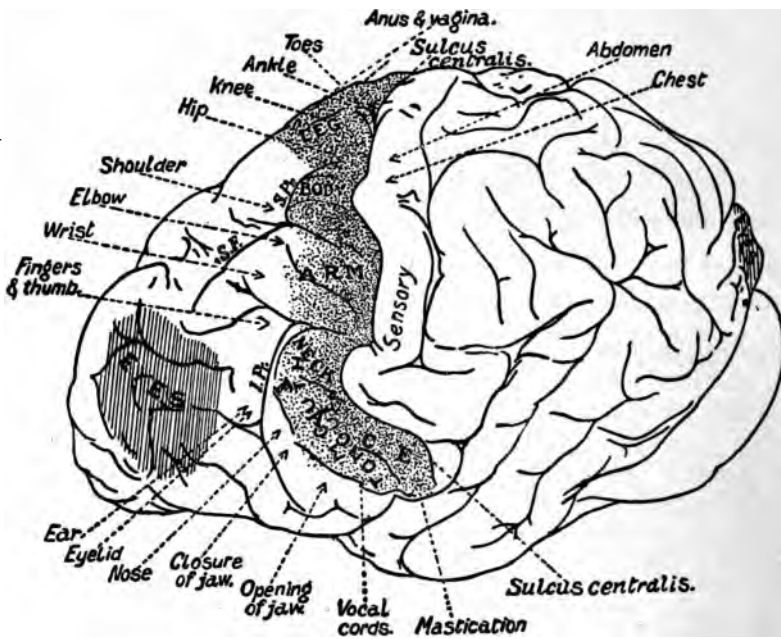


FIG. 22.—Chimpanzee's brain. To show motor and sensory areas of brain cortex. (After Sherrington-Grünbaum.)

which in turn is a section of the movement of the musculature as a whole. The demolition of any part of this functional unit affects the whole to a degree which depends upon the site, the extent and the rapidity of development of the destruction and upon the power of compensation which is inherent to the coördinating mechanism that remains structurally unaltered.

The amount of reflex disturbance is difficult to deter-

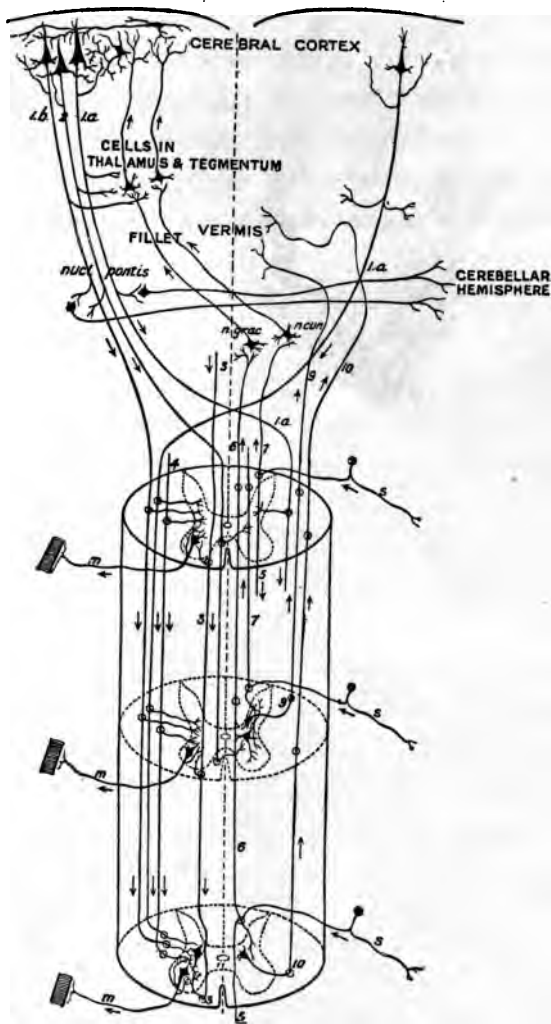


FIG. 23.—Diagram showing the course, origin and termination of the fibers of the principal tracts of the white matter of the spinal cord.

"Descending" tracts: 1a, a fiber of the crossed pyramid or corticospinal tract; 1b, an uncrossed fiber of the pyramid or corticospinal tract passing to the lateral column of the same side; 2, a fiber of the ventral pyramid or corticospinal tract; 3, a fiber of the ventrolateral descending or ponto-spinal tract; 4, a fiber of the rubrospinal tract; 5, a fiber of the comma tract. "Ascending" tracts: 6, a fiber of the dorsomesial spinobulbar tract; 7, fibers of the dorsolateral spinobulbar tract; 9, one belonging to the dorsal spinocerebellar; 10, a fiber of the ventral spinocerebellar tract. (Quain's "Anatomy," published by Longmans, Green and Co.)

mine except in such simple reflexes as the knee-jerk, for movement is voluntary, not reflex. In adult life, we desire a posture and its attainment more or less automatically follows. The desire excites a postural image. The association between a postural image and its reproduction is fixed by the mordant influence of years.

The motor reproduction of the postural image is elicited through the Rolandic area of the cerebral cortex. The pyramidal tract fiber that passes from the cell of the Rolandic area, through the corona radiata, internal capsule, pons and medulla, ends in the motor cell of the anterior horn of the spinal cord or in its homologue among the basal nuclei. The cortical activity is discharged through the axons of the anterior horn cells to evoke muscular movements.

The movements excited at the Rolandic area by electrical stimulation are divided by Sherrington into three groups:

“In one group, movement evoked from the cortex of one hemisphere seems a fraction of the natural movement, the natural movement requiring in its completeness the corroboration of the symmetrical area of the cortex of the opposite hemisphere. In a second group, instanced by conjugate lateral deviation of the eye-balls toward the opposite side, it is equally obvious that the reactions of symmetrical areas of the right and left cortices are related to one another as antagonistic reactions. In a third group of cases, the reactions of symmetrical cortical areas, right and left, seem neutral to one another. Thus, with the area which yields movement of the thumb, that reaction seems neither to reinforce nor to interfere with the similar reaction evoked from the identical area of the opposite hemisphere.”

Thus, cortical motor cells, just as the motor cells of the spinal cord, show reciprocal innervation and the

phenomena which result from it. The motor reactions elicited through the reflex mechanism can be duplicated by stimulation of the cerebral motor cortex. Cerebral and spinal activities are conveyed to the muscles along a common path, the axon of the anterior horn cell, the peripheral motor nerve. Cortical excitation of muscles may, therefore, reinforce reflex excitation. Hence cortical action may compensate in some degree for loss of reflex action, may mask during movement reflex defects.

Before reflex disintegration is visible to the naked eye it may be detected in graphic records of movements, especially of the exaggerated defensive withdrawal of the lower limb which, after delay perhaps, follows scratching of the sole of the tabetic foot.

In such a graphic record may be evident that the antagonists do not adequately relax when the agonists contract. Muscles acting similarly on the same bony lever do not contract synchronously and the wonted con-



FIG. 24.—The three stages in the normal grasp. (Graham Brown.)

sonance of muscles, acting simultaneously on different levers in a complex movement, is lacking. The absence of relaxation in the antagonists may even be palpable. The lack of consonance (asynergia) may be obvious in an affected upper limb on closing the fist: the extension of the hand on the wrist which normally occurs syn-



FIG. 25.—The tabetic grasp showing asynergia: normally in grasping, the hand makes with the forearm an obtuse angle; here the hand and forearm are in the same straight line. (Graham Brown.)

chronously with the contraction of the flexors of the fingers, disappears; the closed fist is no longer bent back on the forearm; fist and forearm are either in the same

straight line or the fist is bent forward (Fig. 25).

In tabes cerebral action may mask reflex defects. Reflex defects are not revealed during voluntary movement until the cerebral control fails: the amount revealed depends on the degree of the failure. The tabetic moves by his brain to the extent necessitated by the reflex disintegration. Defective voluntary movements, ataxic movements, are no index to the degree of the reflex disturbance.

CHAPTER III

RESULTS OF THE SYPHILITIC LESIONS

2. DISTURBANCE OF SENSORY FUNCTION

Course of the sensory impulses from the periphery to consciousness. Specific nature of the impulses conducted by sensory fibers. Sensory images. Factors which determine the fate of the sensory images. The peripheral impulse and the mental reaction to it. Measurement of stimuli. Sensory thresholds. Their variability. Complex content of the sensation which arises from a simple stimulus. Sensory results of a complete and of an incomplete lesion of the posterior root. Selective invasion of the root fibers conducting pain. Touch loss. Temperature loss. Vibration loss. The postural sense. Paths of sensory fibers from the muscular apparatus and from the postural sense organ of the head. Loss of the sense of the body's posture. Measurement of the loss of postural sense. Effect of blindness and of training on postural sense in the non-tabetic and in the tabetic. Variability of postural sense loss. Postural sense loss inaccurate index of the severity of the tabetic lesions.

THE peripheral processes of the spinal ganglion cells terminate in sensory receptors, some of which respond only to touch, others to pain, others again to temperature, still others to pressure. Of the central processes some, therefore, conduct only tactile impulses, others pain impulses, others again temperature impulses, and still others tension impulses. The sensory impulses arising in the peripheral receptors pass by the posterior root fibers to their first cell station in the nuclei of Goll and of Burdach. Then the sentient of these impulses are conveyed by a second relay of fibers to the ventrolateral region of the opposite optic thalamus, their second cell

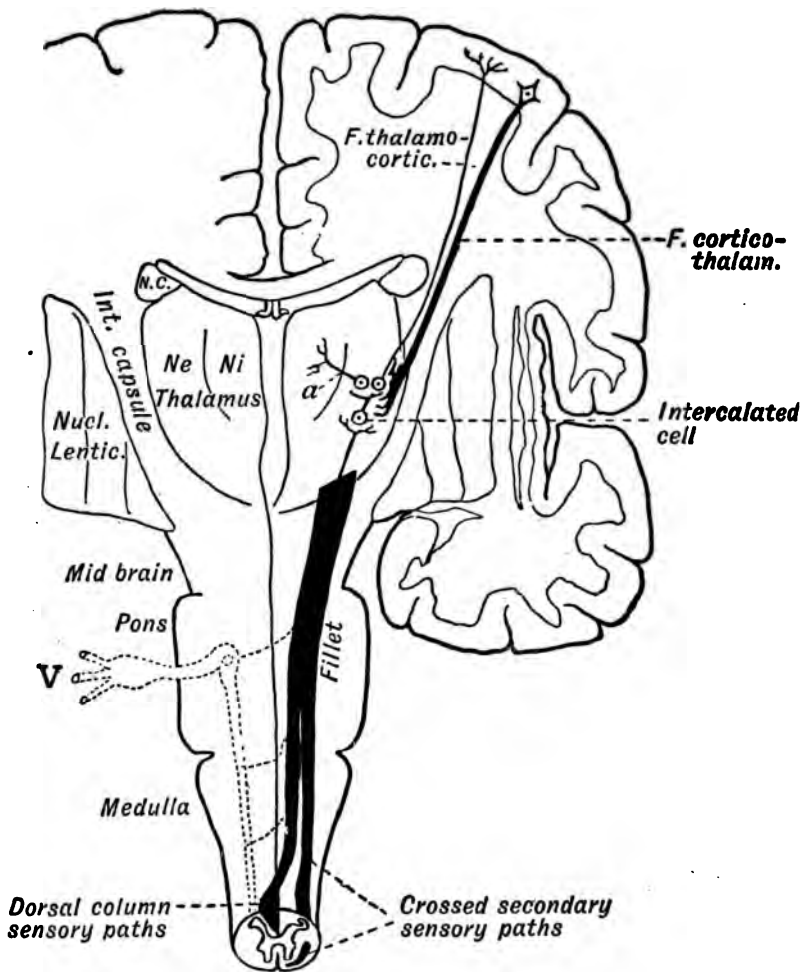


FIG. 26.—To represent diagrammatically the anatomical arrangement of the paths and centers concerned in sensation. Two distinct paths exist in the spinal cord; a crossed secondary path in the ventrolateral column which conveys impressions of pain, temperature and touch, and a second uncrossed path in the dorsal column which also carries touch, and in which run impulses that underlie the sense of position, the appreciation of movement, the discrimination of two points, and the recognition of vibration, size, shape, form, weight and consistence. This second path decussates in the lower part of the medulla oblongata, but runs separate from the first path at least as high as the pons. All these secondary sensory fibers, now crossed, terminate in the ventrolateral region of the optic thalamus. The impressions they carry are regrouped here and, through intercalated neurones, are distributed along two distinct paths; the one carries impressions to the cerebral cortex, the other, we assume, towards the more mesial parts of the optic thalamus. The corticothalamic fibers, which terminate in the lateral nucleus of the optic thalamus, are also shown. (Head and Holmes.)

station. A third relay of fibers conveys them to the cerebral cortex where they excite sensory images. The nature of the exciting impulses these fibers conduct determines the nature of the images excited in the cortex.

The fate of the newly excited sensory images depends chiefly upon their power to command attention. That

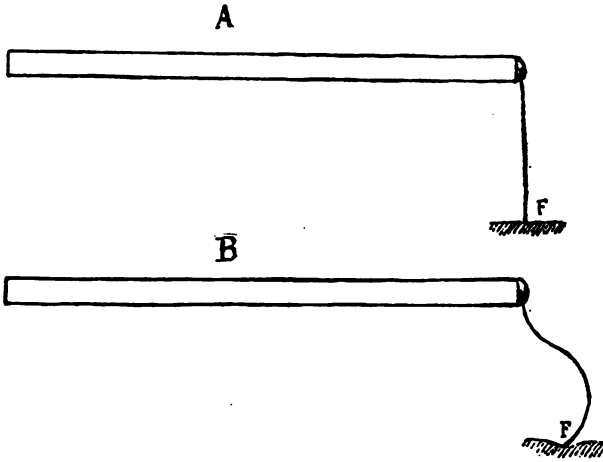


FIG. 27.—Von Frey's Hairs (Barker) affixed with sealing wax to wooden handles; A, the hair applied to the skin surface, F (F should show dimpling as in B). With every hair a maximum pressure may be applied. Beyond that maximum the hair bends. B shows the effect of attempting to apply a pressure greater than the force with which the hair can resist bending.

power increases with their profusion and with their intensity. Whether a sensory demand for attention be or be not successful, depends not only upon the power of that demand but also upon the power of other and simultaneous demands and upon the available store of attention.

The amount of attention which the newly excited images gain is devoted to associating them with analogous images of past experiences that are stored in memory. By the gained amount of attention, the association of the present images with the memory images of

the same order is facilitated and the association of all sensory images alien to that order is inhibited. If the facilitation be adequate, the sensory images excited by the peripheral stimuli evoke a sensation, the stimuli are perceived. If the facilitation be inadequate, no sensation arises, the newly excited images are unconsciously suppressed, the stimuli are ignored.

By instruments which apply measured touch (von Frey's hairs), pain (algometer), pressure touch (aes-

Number by which the hair is known	Pressure in grammes	Measured radii μ	Total area in mm. ²	Radius of a circle of the same area in μ	Pressure per unit area	Tension
8	0.04	30 × 54	0.005	40	8 grm./mm. ²	1 grm./mm.
12	0.1	47.5 × 57.5	0.0085	52	12 grm./mm. ²	2 grm./mm.
14	0.21	55 × 90	0.015	70	14 grm./mm. ²	3 grm./mm.
21	0.23	40 × 80	0.011	58	21 grm./mm. ²	4 grm./mm.
21	0.36	60 × 90	0.017	73.5	21 grm./mm. ²	5 grm./mm.
23	0.88	100 × 120	0.0377	110	23 grm./mm. ²	8 grm./mm.
35	1.4	100 × 130	0.041	114	35 grm./mm. ²	12 grm./mm.
40	1.8	115 × 125	0.045	120	40 grm./mm. ²	15 grm./mm.
70	3	115 × 115	0.042	115	70 grm./mm. ²	26 grm./mm.
90	3.6	100 × 130	0.041	114	90 grm./mm. ²	32 grm./mm.
100	3.5	80 × 140	0.035	110	100 grm./mm. ²	32 grm./mm.
110	4.8	105 × 130	0.044	120	110 grm./mm. ²	40 grm./mm.

FIG. 28.—Table of von Frey's hairs. (Head and Holmes.)

thesiometer), pressure pain (algometer), or other stimuli, we can ascertain the minimal stimulus of a given order, which at a given site, can evoke a sensation. This minimal value marks the threshold below which stimuli are not, as a rule, perceived.

When, say, touch is tested in an attentive person, if subliminal stimuli be first applied, delusions of touch may arise and may persist, until, with an adequate stimulus, an unimpeachable sensation of touch is evoked which affords a criterion for further judgment. When stimuli are applied in succession from a normal to a contiguous area of sensory loss, the extent of the area of sensory loss is smaller than when the stimuli march from the insensitive to the normal area. A positive response

may thus be given to an imperceptible stimulus and a negative response to a perceptible stimulus. The response elicited is the resultant of two factors, the peripheral stimulus and the mental reaction.

No sensory threshold is constant: it varies as the attention ebbs and flows. It is higher in fatigue than in freshness, in depression than in cheerfulness, in apathy than in interest. The simpler the perception, the less evident is this oscillation; the more complex, the more evident. Hence, the simplest sensations evoked by peripheral stimuli serve best as a guide to the integrity of the conducting paths. Complex sensations, such as are involved in weight appreciations, are more suited for mental tests than for sensory.

Between successive sensory perceptions there are three measurable variables: the rate, the direction, and the extent of the change. As a rule, only the last is clinically used. The average minimal perceptible stimulus in all mental states is taken as the sensory threshold. The extent of the sensory loss is that revealed

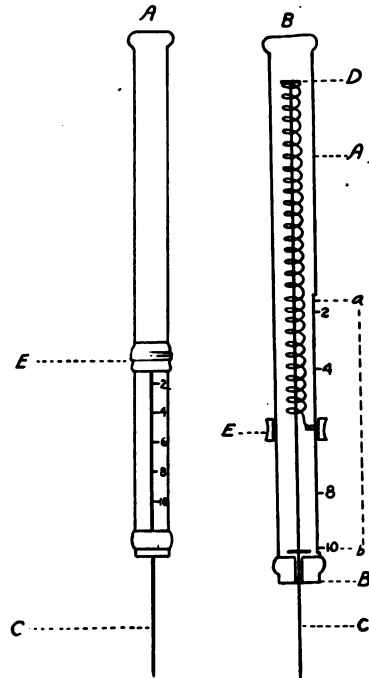


FIG. 29. — Algesimeter. (Head and Holmes.) By moving the regulator, *E*, the tension of the spring can be increased or diminished to an extent measurable on the scale, *a*—*b*. The penetrating force of the needle point, *C*, necessary to elicit pain, can thus be approximately ascertained in terms of this arbitrary graduation.

when testing only from the normal to the anaesthetic area. The distribution of the sensory loss indicates the locality and extent of the lesions.

Touch stimuli are supposed to differ from pressure stimuli in that the latter deform the skin surface. Every touch deforms the skin. Von Frey adopted arbitrary

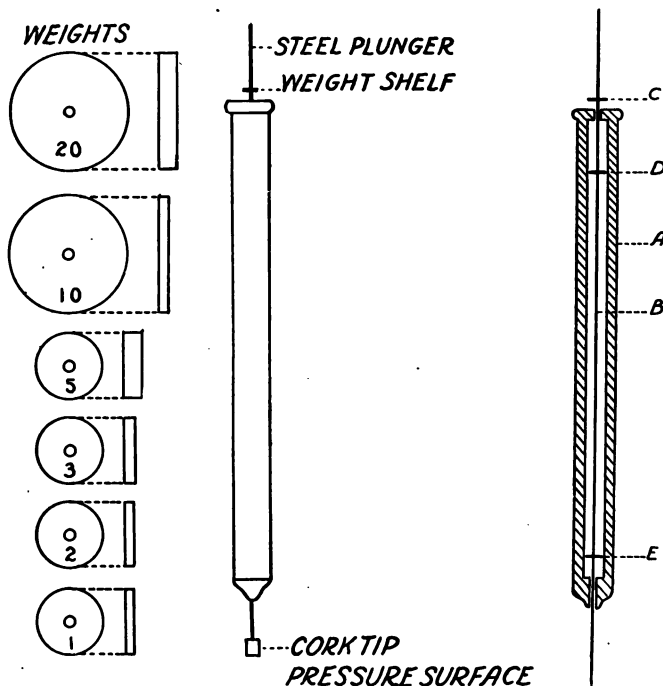


FIG. 30.—Pressure esthesiometer (Holmes) for testing pressure touch. By means of the gram weights, threaded upon the steel plunger *B*, resting on the weight shelf *C*, the pressure on the unit area of the cork tip which is applied to the skin can be varied.

standards to define touch from pressure. Pressure beyond a certain power elicits pain. Pain induced by pinprick implies a touch. A temperature stimulus has usually a touch and, if of sufficiently high or low degree, a pain element also. The sensation contains not only the character of all the specific impulses which evoke it, but

VIA SELL

also that of the site stimulated. Both the nature and the localization of the stimuli are perceived.

If the syphilitic lesion completely severs the posterior root, all the impulses which the affected fibers convey from their sensory receptors are entirely blocked. No impulses pass the lesion; no impulses reach the corresponding cells of the synapses at the nuclei of Goll and of Burdach; no impulses pass thence to the cortex; no images are excited; no sensations are evoked, however much the sensory receptors of the destroyed root be stimulated. A lesion in the first of these three relays of fibers which are linked in a common function is thus physiologically equivalent to a lesion of all three relays.

The lesion in the posterior root, if incomplete, seldom affects the various sensory groups synchronously or equally; thus, the pain loss is, as a rule, the first, the most extensive and the most severe. The syphilitic

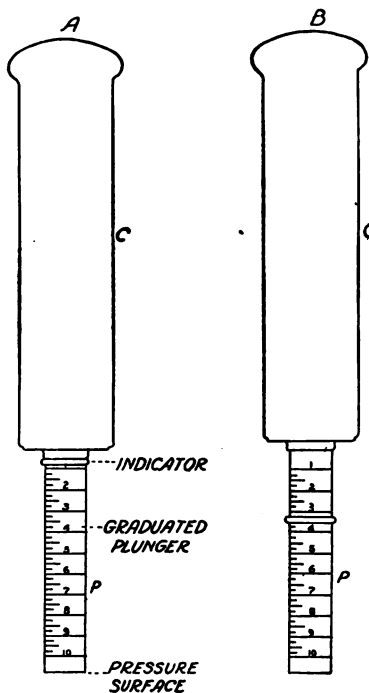


FIG. 31.—Modified Cattell algometer for measuring pressure pain. The pressure surface is applied to the supported investigated area and pressure is exerted upon the knobbed end. As the pressure increases, the plunger, *P*, is pushed up into the case while the indicator is forced down the scale. After the pressure has been removed from a part in which a pressure of $3\frac{1}{2}$ kilos per unit area produced pain, the position of the indicator would be that shown in *B*. The algometer may be obtained graduated in kilos or in pounds.

lesion in the posterior root tends to affect the root fibers in functional groups and within these groups to implicate the fibers unequally. Some fibers may be completely severed, some may escape, some may be partially destroyed. As a rule, the axis cylinder persists, but it may shrink and undergo microscopic changes which we little understand. The myelin sheath degenerates. It is said both to isolate the axis cylinder, so that the impulses conveyed may be confined to their course; and to increase the conductivity of the nerve. But nerve impulses encounter resistance mainly at the synapse. The resistance at the synapse is high in conditions of defective nutrition of the neurons. Resistance is notably raised at the synapse of a nerve process partially severed by a syphilitic lesion in the posterior root from its food supply, the ganglion cell. The more complete the severance, the higher is the resistance, the higher the sensory threshold.

The passage of impulses is swifter along the nerve fiber than through the synapse. The higher the resistance at the synapse, the more is the delay in passing there. Delayed perception, which is frequently seen in tabetics, is a sign of an incomplete lesion. As a phenomenon of an incomplete lesion we must add to a raised threshold and delayed perception a sensation of imperfect content. Thus, touch in tabes may be perceived, and yet be imperfectly localized; pinprick may be felt as a touch without any pain element; pressures almost injurious to the tissues may induce no pain; a burn may provoke merely a sensation of warmth.

In tabetics, areas of complete insensitivity, especially to pain, or of diminished, delayed and imperfect sensitivity occur. Such areas are encountered usually first on the lower limbs; next, most frequently, on the thorax;

then, on the ulnar side of the arms; then on the remainder of the trunk; and last on the head and neck and face. In the lower limbs the area of the fifth lumbar and first sacral roots is usually first affected; on the thorax, the breast. But the sensory loss may exceptionally begin at other sites and may predominate there:

Among the fibers conducting impulses of the same order, the long are usually first and most severely implicated. Thus, the sensory loss, as a rule, is greater over the feet and hands than over the less distal parts of limbs, and greater over the front than over the back of the thorax.

The loss of tactile sensibility usually has an evident root distribution. Associated with this loss are troublesome paraesthesias, sensations of insects crawling upon the skin, of tingling and of numbness. The patient may feel as if he walked on rubber or on felt or on cotton wool. Similar sensations are experienced in walking after cocainization of the skin of the soles of the feet.

Preceding tactile loss, loss of sensibility to pinprick invariably occurs. Sites which show at first merely a blunting of pain sensibility may speedily become quite analgesic. There may be delay in perceiving pain stimuli, then a sudden exaggerated response, followed by unwonted persistence of the painful impression. The loss may be total; the strongest stimulus of the algometer may be perceived not at all, or merely as a touch.

From the temperature sense not only the pain and the touch but also the essential thermal element may disappear. On skin areas where a change of three to five degrees may normally be appreciated, twenty or thirty degrees of difference may pass undetected. Usually the tabetic can tell a change has occurred before he can

detect whether it is hotter or colder. Associated with temperature loss, sensations of burning or of sudden trickling of cold or of hot water may arise in the limbs.



FIG. 32.—Muscle spindle. (Dogiel, Graham Brown.)

Egger and Déjerine have emphasized the loss of vibration sense in tabes. This loss affects simultaneously



FIG. 33.—Tendon organ. (Dogiel, Graham Brown.)

the bones, muscles and subcutaneous tissues, and may be coextensive with the loss of pain.

The sensory receptors in muscles, tendons, ligaments

and cartilage are attuned to respond to changes in the muscular apparatus. Sentient impulses from these receptors pass along the common afferent nerve through the posterior roots and thence by the sensory relays to the cerebral cortex. The sensory receptors in the labyrinth of the ear (page 30) are attuned to respond to change of the planes of the head. Sentient impulses travel from these receptors by the vestibular nerve to the vestibular nucleus, thence to the optic thalamus and finally to the cortex.

Sentient impulses from the muscular apparatus and from the labyrinthine organ are constantly exciting in the cerebral cortex kaleidoscopic postural images of the whole body, always changing, yet always related. Of the majority of these images we are unconscious. Were we

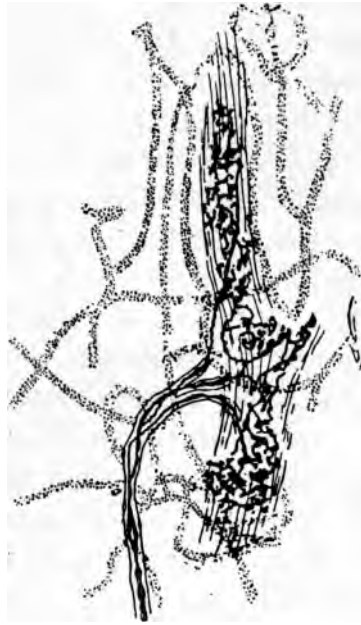


FIG. 34.—Ruffini's corpuscle, from ligament of knee. (Sfameni, Graham Brown.)

constantly aware of our posture, we could not think unless we were absolutely at rest. Even when we are concentrating attention upon postural images, we are conscious neither of all the postural images nor of all the changes in them. Only the most persistent, the final, or the intrinsically dominant of the postural images, or the resultant from a series of successively evoked images, or from a group of such series, may reach con-

sciousness. We are usually conscious, not of the postures of the various parts of the body, but only of their combined effect, of our relation to our environment. In this combined effect, the importance of the several parts varies according to the posture; but except in the recumbent posture the attitude of the head dominates our conception of the body's posture.

The lesion of the posterior root may partially or completely interrupt the sensory impulses underlying appreciation of position in any section of the body. Lesions in the head segment, labyrinthine affections, are more disturbing than lesions elsewhere and lesions of the trunk than lesions of the limbs, and lesions of the lower than those of the upper limbs.

Loss of the sense of the body's posture has not been measured. The gross content of this relation is sometimes so distorted that tabetics, when recumbent and at rest, feel as if they were rotating; and when erect, as if they were reeling or falling.

Loss of the sense of posture in a section of the body, a limb or a part of a limb, has been estimated by duplicating with its fellow, reproducing on a mannikin, or describing, the position and attitude of the investigated section. An existing posture is changed to a new one through a series of intermediate postures. Most clinicians use the appreciation of this change as an index to the power of appreciating posture, an index to the so-called postural sense.

Goldscheider showed that with a constant rate a certain minimal extent of passive movement is normally needful before a postural change can be detected. This extent varies in different joints; the threshold for large joints is lower than for small. The threshold is not of constant

value. In testing a finger joint, movements through an arc of 5° , of 6° , and of 4° , may be respectively necessary to elicit a response in three successive trials. The thresh-

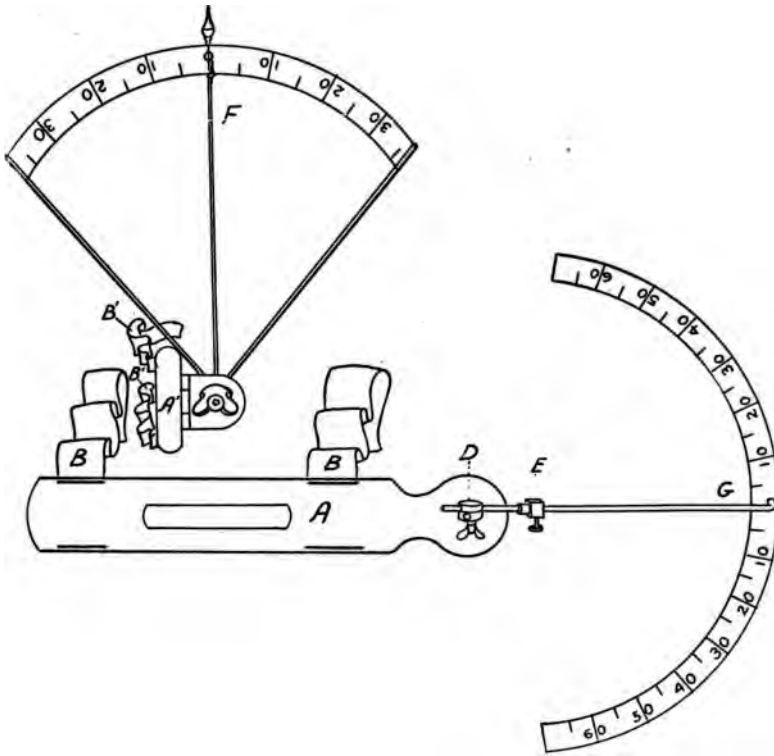


FIG. 35.—Goldscheider's instrument (model used by Head and Holmes) for measuring joint movements. By means of the bands, *B*, the metal plate, *A*, is immovably attached to the limb, proximal to the moving joint, and in the plane of the movement: Into the slot, *D*, the scale is inserted. The joints at *D* and *E* permit the scale to be rotated into any plane without detaching the plate *A*.¹

old varies with the attention, but its average value, 5° , is practically constant in the same joint in the same individual. Simultaneously with the appreciation of

¹ These instruments for measuring sensation can be obtained from the Dressler-Beard Mfg. Co., New York City.

movement comes ordinarily the appreciation of direction; both normally have the same threshold.

The syphilitic lesion of the posterior roots, if completely severing the fibers conveying the impulses underlying postural sense, entirely abolishes perception of posture in a joint. If the severance be incomplete, the greater the destruction is, the feebler, more delayed and more sparse are the impulses which reach the cortex to excite postural images; and the higher is the threshold for perception of movement. Movement, if appreciable at all, must be of abnormally great extent before it can be detected: then its direction still remains unknown. The movement may need to be increased fifty per cent or more before the direction is discerned. The threshold of direction is higher than the threshold of movement.

The lower limbs are more often affected than the upper; the head rarely escapes. There may be complete loss of postural sense in the toes. The ankle is more affected than the knee, the hip less than either. Similarly the shoulder is less and the finger more affected than the elbow or wrist. Loss in the large joints never rivals that in the small.

After a sensation of a given order has been evoked by a perceptible stimulus, before a change can be perceived, the intensity of a superadded stimulus must bear a certain ratio to that of the existing stimulus. The amount of change necessary for detection varies with the sensory order: a change of one eightieth to one hundredth in the intensity of visual stimuli can be detected: of about one-seventh in auditory stimuli. Although such average values may be determined, the threshold of perception of change presents individual variations of considerable extent. Musicians may be

able to detect tone differences which are imperceptible to the untutored ear. Slinger and Horsley, reporting their investigations of the muscular (postural) sense among the students of a college for the blind, state:—

“ . . . if the information gained by sight is permanently blotted out, the muscular sense under necessity can by education be brought to a point at least one-fourth better than that learnt by the normal seeing individual. In confirmation of this conclusion, the plate test showed in a very striking manner that the pupils who were completing the technical training of the college were notably superior to those who were just entering on their special education.”

Blind tabetics possess a remarkable freedom from postural sense loss. This freedom is in part due to the localization of the lesions, but in a tabetic who had lately become blind I was able to measure the subsequent improvement in the postural sense. Förster, comparing a group of blind with a group of seeing tabetics, found loss of touch, a perception demanding little discrimination, approximately equal in both groups: whereas postural sense appreciation was faulty in eight of the seeing but in only four of the blind.

Balancers and jugglers and others who specialize in movement have more acute discrimination of posture than ordinary individuals. By the courtesy of Dr. Wachsmann, Director of the Montefiore Home, I demonstrated to the students of the International Extension Course at Fordham University in 1912, a patient who was a professional skater, who for over seven years had had nearly every tabetic sign, but only insignificant postural loss. I have treated three sailors, one officer and two common seamen, who were long tabetic, and who

showed pain, tactile and pressure loss, but only slight postural sense loss.

By psychotherapy, the postural sense can invariably be improved, sometimes to a miraculous extent. The improvement occurs without any apparent change in the lesions. I observed it occur once in spite of an advance in the lesions. It is partially independent of the lesions. The postural thresholds, therefore, seldom indicate with precision the severity of the structural change.

CHAPTER IV

RESULTS OF THE SYPHILITIC LESIONS

3. DISTURBANCES OF ATTITUDE AND MOVEMENT

The changing and maintaining of attitude. Attitude expressive of the motor tendencies that we desire and of those that are unconsciously evoked by the mental state. The impossibility of maintaining an immovable posture. Normal swaying. Tabetic swaying. Romberg's sign. The tabetic attitude. Effect of blindness, psychotherapy and mental states on the tabetic attitude. The tabetic attitude a consequence not only of the lesions but also of the mental state. Ataxic movements. Controversy regarding the cause of ataxia. Importance of the constituent parts of the muscular apparatus in coördination. Postural sense loss not parallel to ataxia. Reflex defect not necessarily associated with ataxia. Onset and course of ataxia. Fatigue, fear and shock as precipitants of ataxia. The ataxic moment. The relation of ataxia to the tabetic lesions. Psychic control of coördination. The ratio between effort and accomplishment in movement. Voluntary movement the resultant of the desired and of the unconsciously evoked postural images. The interaction of voluntary and of reflex movement. Psychic activity masking reflex defect in voluntary movement throughout the preataxic stage. Ataxic complexes. Coefficient of ataxia. The reciprocal relations of ataxia and the mental state. The amount of disturbance of attitude and movement mainly a measure not of the structural changes but of the degree of the failure of conscious control of movement to compensate for the tabetic defect in the reflex mechanism. The surrender or so-called "paralytic" stage.

IN infancy, we consciously acquire power to alter the relation of the various bony levers of the skeleton to one another and to the horizon. We learn first to hold up our heads, then to sit erect, and last to stand. In adult life, if we merely desire to change our attitude, say,

from sitting to the erect posture, the act automatically follows.

In maintaining an attitude, we conserve the position of the skeletal levers against a constant force acting in a constant direction, the force of gravity. The tone of the muscles whose action is opposed to that force is kept high that these muscles may remain taut; and the tone of their antagonists low, that those may be lax. The position of the head reflexly determines that of the rest of the body.

The posture does not exclusively reflect the desire. The mood, the emotion, the mental state at the moment it is assumed, have motor attributes of which we are unconscious, but with which the desired posture must be compounded. The influence of the mental state on posture is universally recognized; everyone can distinguish the attitude of the tired from that of the fresh; of the apathetic from the alert; of the depressed from the joyous. Indeed, mental states are often defined by their resulting attitude: thus, the sad droop; the craven are "spineless" and "weak-kneed"; the brave are upright; and so forth. The posture attained is the resultant of both the desired and the unconscious motor tendencies.

Even with an absolutely normal motor mechanism, we cannot maintain an immovable posture. Soldiers and others may be trained to stand with the feet alongside and parallel to one another, apparently motionless, but a writing point fixed to the head of the most steady, invariably records fourteen to sixteen oscillations per second; and if the investigated person be warned to expect a sound from the right or from the left, the oscillations become directed towards the expected sound. The

oscillations are aggravated on closing the eyes and in constrained attitudes, such as standing on one foot, or bending. These oscillations, usually imperceptible to

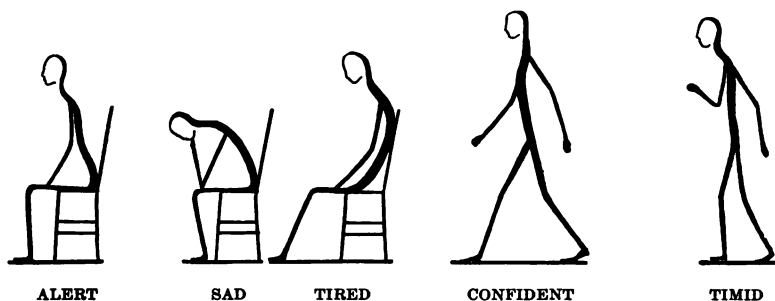


FIG. 36.—“The mood, the emotion, the mental state, at the moment—have motor attributes of which we are unconscious.”

unaided vision, become evident in the tabetic. He sways obviously when he stands with closed eyes (Romberg's sign).

Swaying is first evident in the tabetic only when

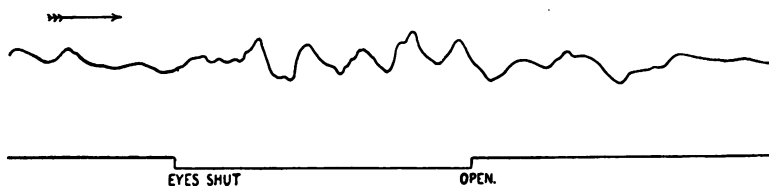


FIG. 37.—Tracing¹ of lateral swaying in a normal person showing the gradual increase of the extent of the swaying after the eyes are closed. (Graham Brown.)

¹The Knauer-Maloney cephalograph records the resultants of the swaying in all planes. I give no examples of its tracings for I find some of the oscillations it records are due to a mechanical defect in the machine, a defect which is easy to remedy but which I have not yet cured. The present type of the machine is of value merely for clinical work but not for scientific. Only the tracings taken by the same observer with the same cephalograph are comparable. Those who may be interested in its design will find it fully described and illustrated in *The Journal of Nervous and Mental Diseases*, Sept., 1914.

he is standing with closed eyes, in constrained attitudes. Under similar circumstances, many who are not tabetic may be seen to sway. There is no limit of non-tabetic instability, no threshold of Rombergism. But usually



FIG. 38.—“To counteract the disturbance of balance, the erect tabetic has recourse first to one stick, the tripod stage” . . . (Montefiore Hospital Case.)



FIG. 39.—“To counteract the disturbance of balance the erect tabetic has recourse—then to two sticks, the quadruped stage.” (Montefiore Hospital Case.)

the non-tabetic instability is slight; develops when the constrained posture is long maintained; and, on successive examinations, does not increase. Tabetic instability is, as a rule, immediately apparent when the constrained posture is assumed, is aggravated by continuance of the posture, and on reëxamination discloses a tendency to increase. This tendency to increase, I think, is the

chief distinction between doubtful tabetic and non-tabetic swaying. Knauer and Maloney devised a cephalograph¹ which graphically records head movements in three planes. By its use, the extent of the patient's swaying is ascertained and recorded when he is first examined. By comparing the first with subsequent records, increase in swaying can be detected. A diagnosis of Rombergism can thus be early reached.

Tabetic swaying, at first obvious only in constrained attitudes, is next evident in normal, then in broad base, and finally in supported, attitudes. It later may occur successively in these attitudes even when the eyes are open. The wider the arc of the swaying, the broader must be the tabetic's base, if he is to remain erect. He may sway through an arc so wide that to enlarge his base he moves his feet; he reels. Reeling on eye closure in the usual erect posture is always pathological.

To counteract this disturbance of balance, the erect tabetic has recourse first to one stick, the tripod stage, and then to two sticks, the quadruped stage. He may ultimately become unable to assume and to maintain



FIG. 40.—Further development of the "quadruped stage." The blurring of the picture was due to the patient's swaying. (Montefiore Hospital Case.)

¹Made by the Medical Machinery Company, Detroit, Michigan.

any attitude except the recumbent. This deterioration of attitude may halt at any stage. The courageous can always maintain, although distortedly, an erect posture. The timid surrender after passing through the tripod and quadruped stages, and become chair-ridden and finally bed-ridden.

The blind tabetic, as a rule, conserves his power to



FIG. 41.—“The courageous can always maintain, although distortedly, an erect posture.” (Montefiore Hospital case.)

stand erect. A tabetic who has lost that power and who becomes blind may then spontaneously regain it. Under the influence of psychotherapy, the recumbent tabetic may be made to rise again and to stand steadily even with closed eyes. Equilibration is more difficult in fear, fatigue and depression, in the seeing than in the blind, in the untrained than in the trained.

If a blindfolded, seated tabetic, whose feet, back and head are well and comfortably supported, attempt to maintain his arms outstretched in front of him, the arms may fall, one, perhaps,

faster than the other. Sometimes the fall is slow and steady. Usually it is somewhat and increasingly unsteady; the arms may oscillate more and more widely, till the tabetic ceases effort and lets them drop. The tabetic to whom the unsteadiness is inappreciable not

rarely is amazed to see his arms fallen, which he supposed were still outstretched.

I have many times tested the appreciation of change of posture at the shoulder, in cases in which spontaneous, unnoticed fall of the arms occurred. For movement at approximately the same rate as the fall, thresholds of less than ten degrees were almost invariable. Indeed, it was usually difficult to move the shoulder of such a tabetic slowly enough to prevent his detecting a change through an arc of more than three or four degrees. A tabetic with a threshold of only a few degrees may be ignorant of a movement through an arc of nearly ninety, of a movement perhaps a thousand per cent greater than his demonstrable postural threshold.

This curious phenomenon is due to the habit which the tabetic acquires of not attending to the postural images which accompany movement. The few feeble and delayed postural impulses, transmitted from the section of the muscular apparatus which corresponds to the diseased fibers in posterior root or in vestibular nerve, excite feeble, imperfect and delayed postural images. Such defective images mislead and delude the tabetic in movement. He sees simultaneously many objects, every one of which confirms his faith in the impeccability of his visual information. He therefore relies on his visual images to judge and guide his movements; and he ignores and suppresses the misleading postural after-images. Hence, unless he consciously concentrates his attention on these neglected after-images, he is unaware of his posture when his eyes are shut. This suppression is not a phenomenon peculiar to postural images. Those who see double suppress the false image; those who are deaf

suppress unintelligible sounds. Suppression of what is undesired is a normal mental reaction.

• Suppression of any image tends to suppress those habitually associated with it. The suppression of the postural images of the outstretched arms tends to suppress also the motor equivalent of those postural images, the muscular action necessary to maintain the posture. The desire to maintain the posture must prevail over the unconscious tendency to suppress it, or the arms fall. The length of the time the posture is maintained affords a measure of the dominance of the desired over the unconscious motor tendencies.

But these unconscious motor tendencies arise also from sources other than the direct suppression of the essential motor images through the suppression of their peripherally excited postural equivalents. The more a tabetic relies on vision the more does absence of vision leave him with a consciousness of sensory perception analogous to that present in night terrors. The lack of peripherally excited postural images from the misty consciousness of light sleep gives rise to the sensation of falling, or to the cognate sense of inability to move; and engenders sudden great fear. The suppression of the imperfect postural after-images by the tabetic, on eye closure, leads to fear. Fear inhibits all associations and, therefore, causes swaying, reeling, and falling. Occasionally, instead of swaying, the tabetic collapses as if suffering from hysterical paralysis. Indeed, fear in the tabetic may be the dominant element in the lack of equilibration. Thus, Dercum reports a case of a blind tabetic negro who could stand steadily so long as his eyes were open, but who reeled and fell when his eyelids closed over his sightless eyes.

But *the* characteristic of voluntary movement in tabes is its disorder, its ataxia. Normal voluntary movements are thrifty in force, accurate in direction, regular in order, uniform in rhythm and precise in extent. Movements lacking this composite quality to a degree evident to the unaided eye are said to be ataxic.

Since ataxia was first recognized, neurologists have conducted researches and controversies upon its cause. Theories of ataxia have succeeded one another like decrees of fashion. Brown-Séquard (1861) taught the spinal or reflex theory. Leyden (1863 et seq.) founded his sensory theory on his observations that ataxia may first appear in a limb where loss of cutaneous sensibility is evident, and that the increase of the ataxia may be roughly parallel to that of this sensory loss. Charcot agreed with Leyden. Erb (1859), Friedreich (1879), and others pointed out that ataxia could occur without loss of common sensibility. Jendrassik (1888) and Raymond (1897) stoutly maintained that neither sensory nor motor, but cerebral causes underlay ataxia. Goldscheider (1889) emphasized the importance of postural sense loss; Frenkel and Förster and others, of loss of tone in the muscles; Déjerine and Egger, of loss of the vibration sense in the bones. Some have cocainized the joints and blamed the loss of sensibility in the articular cartilages; others have removed or cocainized the skin and have absolved it from contributing to ataxia.

In all movement, alterations occur in the pressure at the points of contact on the articular surfaces of the moving joint, and in the tension of the implicated ligaments, tendons and muscles, and of the skin which covers them, and of the bones into which they are inserted. These alterations give rise to stimuli some of which are

sentient, some non-sentient. The sentient stimuli underlie postural appreciation, and with visual impulses afford a basis for comparing the attained with the desired posture. A loss of sensibility in any one of these component parts of the muscular apparatus disturbs postural sense, according to the degree of the loss, and to the power of the sensibility resident in the other parts to compensate for that loss.

Of the component parts of the muscular apparatus, the skin seems to be least indispensable to coördination, for anesthetizing with cocain and even removal of the skin do not greatly disturb coördination. As large joints are more sensitive to movement than small, although a small has a relatively greater articular surface than a large joint, the joint cartilages do not play the major rôle in coördination. The most important element of the muscular apparatus in the function of coördination is the sensory receptors in the muscles and tendons.

In tabes, ataxia may or may not be parallel to the postural sense loss. There may be postural sense loss without evident ataxia. Indeed, such loss is not uncommonly detected during the preataxic stage. The insignificant amount of ataxia seen associated in thalamic lesions with loss of all forms of sensibility, including that of posture, further shows the inadequacy of exclusive sensory theories.

In consequence of the tabetic lesions affecting the non-sentient stimuli, measurable disturbance of the reflex coördinating mechanism results, to a degree depending upon the site, severity, and rapidity of development of the lesions, and upon the compensatory activity of the intact reflexes. The disturbance may by graphic methods be demonstrable before ataxia is apparent, and after

ataxia has been banished by suitable therapy. The reflex theory is, therefore, inadequate to explain ataxia.

The structural changes in the cerebral cortex which Jendrassik described and which he thought substantiated his cerebral theory, have been shown to be not only inconstant but exceptional in tabes.

Yet each of these theories contains part of the truth and all together contain most of it. In tracing the course of tabetic ataxia, I shall emphasize the factors which govern it so that a clear idea of its production may accompany its description.

Ataxia is first noticeable in a limb or limbs, commonly in the lower, not in the upper; but ataxia of the limbs does not occur without incoördination of the head and trunk.

Typically the onset of ataxia is insidious: the tabetic notices he is more easily fatigued; he has difficulty in walking in the dark; he falls into the basin if he closes his eyes while washing; he no longer feels the ground as before; he feels as if he walked on some thick yielding surface, such as rubber, or felt, or cotton wool; he is uncertain in descending stairs or inclines. Uncertainty, at first intermittent, becomes more and more continuous until movement is scarcely possible without meticulous care. This care implies increase of effort; misguided, ill-judged effort causes his uncertainty to grow with every movement. The gait becomes strutting, the movement jerky, chaotic and extravagant; the feet are banged upon the ground; and the trunk is held stiffly at awkward angles.

Sudden exacerbation may occur, which the ataxic attributes to a definite incident, a chill, a fall, a fright, or overwork. After such an exacerbation, improvement

may occur but the ataxia seldom decreases to the degree which existed before the incident happened.

Instead of insidiously progressing the ataxia may advance with fulminating rapidity. Such cases have been especially reported by the French neurologists (*ataxie d'emblée*).

Ataxia may begin suddenly and be from its commencement of severe degree. Gowers (p. 366) records a case in which severe ataxia developed in twenty-four hours. Ataxia may precipitately appear after fatigue. Inglemans reports that Rosalie X, in November, 1891, ran after a tramcar, on reaching which she felt ill. At the end of her journey, when she rose to leave the car, her legs would scarcely support her. She reeled to her home where her ataxia thereafter confined her.

Dr. James Taylor had under his care at the National Hospital, London, a tabetic who, while on duty as a policeman, after a long and swift chase captured a criminal. While the captive was being escorted by others to prison, the tabetic policeman was being carried to the hospital, incapable of effective movement, owing to an explosive onset of ataxia.

Besides fatigue, fear also may act as a precipitant of ataxia. In addition to the following example, I have published another [*Journal of Nervous and Mental Disease*, Vol. 40, No. 11, 1913] and seen a third.

An elderly gentleman decided, not without misgiving, to take for the first time in his life, a short holiday in Europe. He was visiting a cave, which was lit by electricity. The patient told me he did not like the escapade; to go under the earth alone with foreigners seemed to him hazardous. Suddenly all the lights went out. The darkness was absolute. His worst fears seemed about

to be realized. He stretched out a panic-stricken foot for the next step, and felt nothing. Then he collapsed, was gently carried out of the cave and tended in the local hospital. He had completely lost his power of walking. When I saw him two weeks later he had Argyll-Robertson pupils, no knee-jerks, and other classical signs of tabes, together with an incapacitating degree of ataxia.

I have also recorded (*loc. cit. supra*) three cases in which stumbles precipitated the tabetic from coördination into ataxia.

The ataxia in such cases appears suddenly, at a definite, ascertainable moment which we may call the *ataxic moment*. Before the ataxic moment movement is apparently normal; after, chaotic.

In absence of all other ascertainable causes, we must attribute the bizarre onset of the ataxia in these cases to the fatigue or fright or trivial mishap which immediately preceded it. Without this inciting occurrence, the ataxia would presumably have later developed gradually, imperceptibly almost, as is its habit. In cases such as these there is no question of alteration in the lesions. The effect is immediate and entirely mental.

When ataxia develops under observation its first appearance may or may not coincide with a detectable advance in the structural changes. With the progress of the ataxia the lesions may, but usually do not, increase. Increase of the lesions may, or may not, give rise to increase of the ataxia. An ataxic under my care lost the right third and fourth lumbar roots following a salvarsan injection. Before this accident I had measured his postural sense in the right lower limb and had taken several records of his gait. No detectable change happened after the accident.

Daily variations occur: the ataxia is greater in fatigue than in freshness, in despondency than in cheerfulness, in pain than in comfort, in fear than in calm.

Exerted in the form styled reëducation, or as hypnosis, or as Christian Science; or through the medium of mechanical forces such as electricity, or of chemical substances such as salvarsan, or of well advertised fetishes such as rings, or belts, or boots, psychotherapy may decrease the ataxia. The improvement lasts as long as the benign psychic influence is maintained. The degree of improvement may be such as to induce a return to the preataxic stage.

Ataxia may be absent in presence of typical tabetic lesions; an increase in those lesions is not always followed by an increase in the ataxia; without evident alteration in the lesions, ataxia may develop and may progress; under the influence of psychotherapy or blindness, ataxia may retrogress; transient mental states may noticeably increase or diminish the ataxia. Ataxia is, therefore, not wholly dependent upon the tabetic structural changes. It is indubitably a consequence of the tabetic lesions but not an invariable one; it is a consequence to a certain extent indirect and to that extent avoidable and remediable.

A reflex is not subject to such variations. Its constancy and infallibility are among its chief characteristics. Neither does the effectivity of a group of reflexes vary without cause. Hence, the variations in ataxia which result from emotions, moods, psychotherapy and blindness must be due to a component of coördination other than the reflex structural mechanism which some allege is the sole mechanism concerned.

The only influence which is subject to mood, to environ-

ment and to physical conditions, which is greater in the blind than in the seeing and in the trained than in the untrained, is a psychic influence. The efficiency of the reflex coördinating mechanism can be enhanced and diminished during voluntary movement by psychic power.

When we desire to move, we wish to change from an existing to a new, the desired, posture. In the transit we assume intermediate postures which, if we move perfectly, are those that render the movement accurate in direction, and precise in extent.

Normally, in acquiring a new movement, we watch our crude attempts. Watching reduces visual distraction and focusses attention upon the movement. When the movement errs in direction or in extent, we perceive the fault. By concentrating attention upon the faultless postures, we facilitate associations among them and suppress the associations of erring postures we may inadvertently attain. Thus, we learn to assume in succession the optimal postures between the existing and the ultimate posture desired.

The more often the same postural images are linked together the more inevitable is their association. Practice engraves these optimal postures and their sequence on memory. After an interval which varies with the individual and with the complexity of the movement, the eliciting of the desired in association with the existing postural images excites in series the images which link them and suppresses all images alien to the series.

Practice decreases the need for conscious attention; visual fixation becomes, therefore, less and less necessary. Practice lessens the need for correction; visual control, therefore, becomes superfluous. Finally, vision is dispensed with in the performance of the movement

and remains only as a potential critic of the completed act. The amount of visual direction which a movement requires is an index of the amount of attention, of mental effort, it involves. Moreover, gross movements such as walking, so long as they need effort, so long as they are visually directed, are clumsy.

The ratio between the needed effort and the accomplished movement varies as the mental state. In unfavorable states, effort may be spared at the expense of the movement; or, in spite of maximal effort, movement may be imperfect. Thus, movements are slow in fatigue, in depression and in fear; and beside the rate, the strength diminishes and, especially in fear, the rhythm suffers. As the musculature is an organ of expression, of adaptation, mental states and emotions may excite and inhibit postural images; thus, the process of thought in Gallic races is accompanied by gesticulations, often more eloquent than words; and, in the traditional snail-like progress of boys on their way to school, inhibition is evident.

In a voluntary movement the psychic resultant of the desired and of the unconsciously provoked postural images excites the motor cells of the Rolandic area in the cerebral cortex. The effect of this excitation is conveyed by the pyramidal tract fiber to motor cells in the cord, or medulla, or pons. These motor cells we considered first as the final cell stations of motor reflex activity. Upon these final common cells, psychic and reflex influences act conjointly, affecting them in similar ways (page 43). The result of this conjoint action, the motor energy liberated in the cell, passes by a single path, the axon of the final common cell, to voluntary muscle, there to ex-

cite changes which externalize the reflex and psychic activity as muscular action.

In tabes, throughout the preataxic stage, the desire to move excites images adequately to procure orderly movement: the intensity of the excitation of the motor cortex in the brain compensates for faulty reflex action in the cord. The greater the lesions, the more effort is needed to maintain this compensation; *ceteris paribus*, the sooner ataxia develops: ataxia first appears in that one of a pair of limbs which shows the most advanced lesions. The less the initial mental capacity, the more powerful is the ataxic tendency of the lesions; the more trivial is the defect which is capable of causing ataxia; and the shorter is the preataxic stage. The less automatic a movement is, the more effort is needed for its accomplishment, and the less it tends to persist coördinate. Thus the visually directed arm movements are more easily and more speedily decomposed than the automatic movements of walking. Walking is more difficult on stairs or on inclines than on a level surface. In the order of the effort required for its performance under different circumstances, walking becomes faulty.

As fatigue and fear develop, greater and greater effort is needed to evoke with sufficient intensity the desired postural images so that they will dominate the unconscious motor tendencies and will stimulate the motor cortex to a degree which compensates for the incoördinating tendencies of the reflex mechanism.

All the daily influences which contribute to fear, fatigue, and other unfavorable mental states, are influences prejudicial to movement and constitute so many incentives to ataxia, so many ataxic complexes.

The liability to ataxia may be expressed by the ratio of all the ataxic tendencies to the coördinating power.

$$\text{Coefficient of ataxia} = \frac{\text{Ataxic tendencies}}{\text{Coördinating power.}}$$

Of these tendencies, the lesions may practically be considered as a constant: the ataxic complexes, as a variable whose value determines the appearance and the degree of ataxia. The coördinating power depends upon the integrity of the reflex and cerebral mechanisms of movement and upon the effectiveness of the psychic control over these mechanisms.

So long as the denominator of this formula is greater than the numerator, that is, so long as the coefficient of ataxia is less than unity, so long endures the preataxic stage; so long is movement coördinate; so long is repression of the ataxic tendency adequate. When the numerator and the denominator are equal, the coefficient of ataxia is unity; and the ataxic threshold is almost reached. To this value the coefficient had unsuspectedly attained, just previous to the fatigue, stumbles and frights which at the ataxic moment precipitated the cases of acute onset I mentioned (pp. 72 and 73), from coördination to ataxia.

Preataxic tabetics fatigue often and readily, and fear a thousand times a day; blind tabetics stumble in their blindness more than any other preataxic tabetic; yet, until the ataxic moment, ataxia does not develop. In all previous fatigue, frights and stumbles, the tabetic had not reached the ataxic threshold, the coefficient was safely below unity; and any increase in its value which then resulted was inadequate to exert a visible effect upon coördination. The nature and intensity of the occurrence which incites to ataxia may in part determine

its effect, but the main determinant is the mental state at the ataxic moment. Upon the mental state depends the degree of the shock which the occurrence induces. The integrity of the coördinating mechanism determines the power of the shock to precipitate ataxia.

So long as the desired, the consciously evoked postural images, can dominate the unconscious; and so long as this dominance is adequate to excite through the cells of the Rolandic area, a reproduction of the desired postures strong enough to mask the reflex defect of tabes, movement remains coördinate in spite of that reflex defect.

We have seen that the impulses arising from the muscular apparatus during a movement excite images of posture in the cerebral cortex. The resultant of these peripherally excited postural images we may consider as the peripheral reflection or after-image of the movement. There recurs (perseverates) in the brain, after a movement has been accomplished, a feeble image of the conscious desire which called it forth, an image which we may consider as the central after-image of the movement. These after-images are identified in the sphere of the associations. Identification is the minimum association any image may experience. Neither survives nor enters consciousness. The central and peripheral after-images completely suppress or neutralize one another, not, however, wholly without result. From their mutual suppression, a more or less conscious feeling of content, of balance, of proper accomplishment ensues: the critique of the movement is satisfied. If the mutual suppression be not complete, if the peripheral cannot neutralize the central after image, the critique is dissatisfied. Then the movement is at once reviewed to trace the cause

of the uneasiness, the uncertainty, which it has aroused.

The feeble, delayed and imperfect postural images which arise from the musculature in the area of the invaded roots, are inadequate to neutralize the central after-images to which they should be equivalent. The tabetic's vision informs him that in spite of his uncertainty the movement seems perfect. The defective peripheral after-images perplex and distract him. He must constantly reassure himself visually that his movements are not as faulty as they feel. Hence, he relies more and more upon vision to tell him how he moves, and he obviates some of his uncertainty by suppressing the false information from his peripheral after-images of movement.

During this stage of replacing postural images (after-images of movement) by visual images as criteria of movement, of substituting a conscious critique for one which was chiefly unconscious, the tabetic experiences added effort in moving. This greater effort is not wholly attributable to the constant watch he now maintains. Visual substitution is accompanied by conscious suppression of the misleading peripherally excited postural images, the after images of movement. Their suppression tends to suppress all their motor equivalents, because the suppression of any one of a series of images habitually associated tends to suppress the whole series, together with the associations of the series. To overcome this unconscious endeavor to suppress the movement, greater effort is needed to perform it. The fatigue thus entailed is redoubled by the inhibitory influence exerted on movement through the fear induced by increasing vigilance and by growing uncertainty.

When vision has been substituted for the misleading

postural images and the mental state has deteriorated beyond the ataxic threshold, the tabetic feels an extraordinary effort is needed to move, but the amount of that effort he has no means of gauging. He overestimates it. For simple movements, he exerts maximal efforts. Maximal efforts not merely fatigue him, they also lead to a sacrifice of accuracy in direction and in extent. His endeavors visually to guide the clumsy movement make it still more clumsy. With exaggerated force he starts to move. The limb jerks forward, at first, in the desired direction, but its momentum carries it onward in false paths. He sees his error, arrests the movement; over-corrects the error; stops again; jerks onward once more; and so by a zigzag course reaches his object only to overshoot it. His movement thus errs mainly through his imperfect conscious control of it, mainly, therefore, in its spatial qualities.

In extreme cases the effort needed to initiate movement may be more than the tabetic can command; in spite of his wish to move, he remains stationary. Or if he manages to make the effort the slightest additional demand upon his capacity proves "a last straw"; a crack in the floor, a change of level, encountered while he is moving brings his movement to a humiliating end.

I well remember my chagrin when a tabetic, whom I brought before my class in the Post-Graduate Medical School, in June, 1913, to demonstrate the cure of his ataxia, and who unaided could walk excellently, blind-folded, began his effort before the class by punctiliously arranging his surroundings, moving a chair and lifting a tiny piece of paper from the floor, by adjusting his garments, by rubbing his moist hands, and by ceremoniously preparing himself for a great endeavor. After

two or three sudden steps he collapsed in a heap opposite an imperceptible crack in the floor. Then, asked to stand, he swayed and fell, or collapsed without swaying. Supported by my little finger, which he rigidly clutched, he circled freely round the amphitheater. The aid rendered him was altogether disproportionate to its effect. Another tabetic, at present under my care at the Neurological Hospital, Blackwell's Island, refuses to walk; and when coerced he shuffles slowly and extremely cautiously along, supporting himself on both sides by aid of the wall, the bedstead, chairs or table; and collapsing or threatening to collapse at frequent intervals.

After the effort of starting, the tabetic feels to some extent that not he but "It" moves. All tabetics have difficulty in arresting movement; they overshoot their mark: and if, while walking, they are suddenly ordered to halt, they fall or sway widely. In Déjerine's service in the Salpêtrière, in 1906, I saw a tabetic who was projected into a run by the effort needed to start movement and who could stop only by grace of an obstacle.

Every influence which conduces to fatigue, depression or fear conduces to ataxia; every influence which rests, reassures and cheers, alleviates ataxia. Unfavorable mental states beget ataxia and ataxia engenders unfavorable mental states. Tabetics are ataxic because they are tired, afraid, and depressed and tabetics are tired, afraid, and depressed because they are ataxic. The ataxic tabetic reflects his fatigue and freshness, fear and courage, cheerfulness and depression, joy, sorrow, excitement, placidity, anger, pleasure and other psychic states by alterations in the degree of ataxia; ataxia becomes his most eloquent mode of expression. The lessening of ataxia during favorable mental states, such as

freshness, cheerfulness and calmness; and the increase of ataxia during unfavorable mental states are merely phenomena of these mental states.

Without the conscious sensory defect of tabes, there would be no uncertainty; no suppression of the critical after-images of posture; no clumsy attempts to direct movement by vision; no meticulous care; no disproportionate effort; no extravagant movement; and perplexity, anxiety, fear and fatigue would not disintegrate the mental capacity of the tabetic, would not create in him motor tendencies antagonistic to voluntary control of movement. Without the reflex defect this loss of voluntary control would not result in ataxia. So long as desire to maintain or to assume a posture can excite postural images with sufficient intensity to compensate for the existing reflex defects, the tabetic moves coördinately. The disturbance revealed in the tabetic's attitude and movement is mainly a measure, not of the structural defects in the reflex mechanism resulting from the syphilitic invasion, but of the failure of conscious control of movement to compensate for these defects.

Deterioration of movement in the tabetic may not stop at ataxia but may pass into what is erroneously called paralysis. There is no paralytic stage in tabes. An occasional case shows extreme muscular atrophy due to syphilitic implications of the motor neuron, implications which are accidental and not inherent to tabes. Most of the muscular wasting of tabes is not a paralytic, but a disuse, atrophy. Over 90 per cent of the so-called paralytic cases are merely cases which have surrendered the privilege of moving rather than endure the tax which movement entails. Some resign themselves to bed life because with advancing years their capacity for effort

fails, some because of despair following failure to obtain relief through treatment; but most because they will not contend against arthropathies and ataxia, or fractures and ataxia. Some indeed become bed-ridden who were never ataxic (*see* p. 162). I have seen several in whom this surrender stage ensued within a few months of the onset of ataxia. They were remarkable in showing also an abnormally short preataxic stage. The mental inferiority which conduced to the short preataxic period conduced also to the rapid surrender of all voluntary movement. Indeed, one explained she had taken to bed immediately ataxia developed in consequence of the death of her daughter.

The bed-ridden, as a rule, do not remain so. Even without treatment, if they do not develop bedsores, they sooner or later make tentative efforts to move again and oscillate between inactivity and action according to the encouragement to effort they receive. With treatment all can be made to rise and walk. The main difference between the ataxic and the paralytic stage is that in the ataxic stage the tabetic still strives, in the paralytic he has surrendered. The courageous never become bed-ridden. Only the old and the timid seek the shelter of an effortless existence.

The three stages of degradation of voluntary movement in tabes are not stages of structural demolition but stages of mental deterioration. The first or preataxic stage is the stage in which the capacity for effort gradually diminishes until the ability longer to mask the spinal lesions by increased supervision of movement fails; the second or ataxic stage is the stage in which the effects of the lesions are no longer masked but the desire to persist active incites the tabetic to move in

spite of the effort movement entails; the third or paralytic stage is the stage in which the desire is inadequate to encourage the tabetic to the effort necessary to movement.

CHAPTER V

RESULTS OF THE SYPHILITIC INVASION OF CRANIAL NERVES

The olfactory nerve. The optic nerve. Onset, course and nature of optic atrophy. Ophthalmoscopic findings. Psychic and structural factors in visual loss. Effect of mescaline in enhancing vision. Difficulty of measuring visual acuity. Measurement of light perception in nearly blind tabetics. Threshold of perception of light, threshold of perception of change. Blindness; frequency, time of onset, rate of development. Causes of spontaneous improvement in visual acuity. Absence of proof that optic tabes arrests spinal tabes. Influence of blindness upon ataxia. Spontaneous cure of ataxia following blindness. Influence of blindness upon the mental state and upon sensory perception. The relation of loss of vision to coördination. Effect of blindness upon pain and deafness. Third, fourth, and sixth nerves. Diplopia, its causes; squint; spontaneous cure. Means for ascertaining the presence of suppressed visual images. The fifth nerve. The seventh nerve. The eighth nerve. Vestibular lesions. Auditory mechanism. Psychic deafness. Tabetic deafness. Influence of deafness upon the mental state. Increase of deafness with stationary lesions. Other cranial nerves.

THE syphilitic invasion called tabes, although mainly concentrated upon the posterior nerve roots, may begin in, or spread to, other sites, such as the cranial nerves. The cranial nerves may be attacked either at their nuclei of origin, or in their course. The nature of the attack is similar to that described as occurring in the processes of the posterior root fiber: but, owing to the long intracerebral course of many of the cranial nerves, they tend, perhaps more than posterior roots, to become involved in adventitious patches of syphilitic meningitis which they accidentally encounter.

Among the cranial nerves the olfactory, or first nerve, is not rarely affected. The resulting loss of smell is usually bilateral.

When the second, or optic, nerve is implicated, the attack usually occurs early in the disease. When tabes has lasted several years without optic atrophy, optic atrophy seldom supervenes (Pierre Marie).

Rarely only one nerve is attacked. Usually the spirochete invasion begins in one nerve, at a single or at several points; advances for a time; then halts there; and recommences, not in posterior roots or elsewhere, but in the other optic nerve. The destruction of the optic nerve may stop at any stage. Complete degeneration is rare: even in the most severe cases some fibers almost invariably remain unaffected. The invasion may cause only slight destruction. When the optic nerve is the first site attacked, the degeneration usually is more complete than when the optic follow spinal lesions.

Just as in the posterior root, so in the optic nerve, the invasion is essentially primary; the lesions are not due to changes either in the nerve cells or in the meninges. Stargardt found not the retinal cells but the intracranial portion of the optic nerve mainly affected; sometimes lesions occurred in the nerve without any detectable change in the retinal cells; and the changes in the nerve were always more extensive than in the retina. The lesions in the optic nerve also bore no constant ratio to the meningeal changes in site or in severity.

Through the ophthalmoscope the affected nerve is seen as a gray disc with a clearly defined, pigmented, flat rim. The grayness begins on the temporal side. The fundal vessels shrink. Gradually the whole disc may become ivory white, with here and there atrophic re-

mains of blood vessels. This pallor may rarely be unilateral, and is often more marked in one eye than in the other. Accompanying the pallor, contraction of the field and diminution of the acuity of vision occur. As the disease affects mainly the intracranial portion of the nerve, an astonishingly normal looking disc may be associated with considerable loss of vision.

Diminution of vision begins usually in one eye, progresses to a certain point, remains stationary there, and then commences in the other eye. All degrees of loss may be experienced in either or in both eyes. The loss of vision may be insignificant; or it may be such that scarcely form can be detected. Vision, in severe cases, is usually lost to the same degree in both eyes. As the perception of light is a function of the whole retina, and as the optic nerve is practically never completely destroyed, the blind tabetic can almost always distinguish light from darkness. The reading of standard type is usually employed to test for, and to measure, gross changes in visual acuity. To estimate the contraction of the visual field, we use the perimeter. By means of colored wools, loss of perception, first of green, next of red, and then of blue and of yellow, may be found in the tabetic.

By subcutaneous injection of mescaline sulphate, a salt of the alkaloid of the Mexican drug, pelote, the mescal button (*Anhalonium Lewinii*), Dr. A. Knauer and I produced, in ourselves and others, an intoxication in which attention is restricted to one subject at a time. Any subject while under consideration by the intoxicated person occupies conscious attention in a much more intense and exclusive manner than normal.

In testing with this drug a series of eight blind or

nearly blind tabetics, four of them patients of Dr. Wachs-
mann, the distinguished director of the Montefiore Home,
one a patient of Dr. Abrahamson, and three patients of
mine in the Neurological Hospital, I obtained astounding
results. Visual fields enlarged enormously, especially
for red and green. Visual acuity as measured by Snel-
len's type increased greatly; indeed, some were enabled
to read who before injection could not. One tabetic, R.,
who could distinguish little more than light from dark-
ness, when I went to examine him half an hour after
injection, was tossing coins on the floor and stooping
to pick them up without groping. He spent that night
in a fever of excitement, dragging his friends to moving
picture shows that he might, by describing what he saw
on the screen, convince them of the miraculous restora-
tion of his sight. He passed freely through traffic and
entered and left vehicles as do those who see. The effect
persisted for five days; after the second day it declined
rapidly, but during the first three days it was very evi-
dent.

These experiments emphasize how dependent we are
upon what the patient reveals for our knowledge of
visual defects, and also how we may form false conclu-
sions, as to the extent of the optic lesions, from what the
patient imparts to us. Hence, it is not astonishing that
grayness may be present in the optic disc and the vision
remain, apparently, almost normal.

In fifteen tabetics in whom only a trace of vision per-
sisted, I tested the light perception, using standardized
electric lamps of 1, 2, 4, 6, 8, 16, 32, and 64 candle power.
The tabetic was seated in a dark room, at a constant
distance from the source of light, with his back to it,
so as to avoid unequal stimulation of any part of the

retina, and six feet from it, so that he could not perceive the heat changes. The light was turned on and off, not by means of the switch, but noiselessly by turning the bulb in the socket. Each eye was tested separately. The patient was instructed to say "Lighter," "Darker," "Same," "Change," according to what he perceived and, from the beginning to the end of the experiment, to respond without being questioned.

A change in illumination was sometimes confidently and correctly announced after one or two false responses, the perception apparently having been delayed. When this was evident, responses were required at intervals longer than this latent period. The tabetic responded synchronously with every beat of a slow moving metronome. The stimulus was changed at regularly irregular intervals, to preclude guessing. In order that answers based on false, might not obscure those based on true, premises, fifty responses were required in each test. The proportion of correct answers given by the same patient to the same stimulus was liable to vary from day to day. The lowest candle power which on three successive days yielded not less than fifty per cent of correct answers, was taken as the visual threshold.

The equalizing of the retinal stimulation, the elimination of heat and of noise from the light changes, the number of observations, and the repetition of the tests, made the threshold thus obtained an approximately accurate index of the tabetics actual perception of light.

The correctly perceived change was often described as a sudden flash or a sudden shadow, sometimes as a movement in apparently hallucinatory lights and shades. A change was sometimes perceived, and its nature not. There were, therefore, distinguishable two thresholds,

that of perception of light, and that of change. Delusions were evident, particularly near the visual threshold; and there fatigue was quickly produced.

Some of the patients who could tell how many windows were in a ward, how many panes in a window, could describe the appearance of their fellow denizens, and otherwise offer spurious proof of vision, had thresholds of 32 or 64 candle power at a distance six feet from the source of light, thresholds which precluded any appreciation of form.

In periodical examinations, if the number of correct responses progressively diminishes, the conclusion is inevitable that vision is deteriorating. Sometimes the first sign of this deterioration was the rapidity with which fatigue occurred; accurate results were then obtained only in the first ten or twenty responses. After the perception of light was lost, the ability to detect change of illumination still persisted. In one case, with a 32 candle power light the change from light to darkness was not perceived, but that from darkness to light almost invariably was.

The occurrence of blindness in tabes was observed first by Romberg (1841-51). The great Duchenne in his original description of "ataxie locomotrice progressive" mentions two cases associated with blindness. Blindness was thought by the pioneer investigator to be a common phenomenon of tabes. Indeed, Duchenne found optic atrophy in seventeen out of twenty tabetics.

Von Grosz found among one hundred tabetics disease of varying gravity in the optic discs of eighty-eight. In this total he includes congestion, tortuous veins, changes in the color of the disc, any trace of departure from his normal standard. He does not with these findings give

statistics of his observations of similarly trivial abnormalities in the optic disc of average non-syphilitic individuals of the same age as the tabetics he examined. K. Mendel and Tobias found thirty-six cases (8.3 per cent) of severe optic atrophy in four hundred and thirty cases of tabes at all stages. E. Mendel noted twenty-one cases (7.3 per cent) of blindness in two hundred and eighty-eight tabetics. These figures conclusively prove that if syphilis attacks the optic nerve fibers oftener than we suspect, in the vast majority of cases neither blindness nor even a severe degree of atrophy results.

In Martin's twenty-one cases of blindness, the blindness followed the ataxia only once; in Léri's forty, thrice; in Schüpfer's twenty-four, once: in these eighty-five blind tabetics, blindness followed ataxia only five times (5.9 per cent). Syphilitic atrophy beginning in the optic nerve, therefore, much oftener leads to blindness than atrophy affecting the optic nerve after other sites have been attacked.

Among Elschnig's forty cases of tabetic blindness, thirty-one became blind within two years of the first symptom of affection of the optic nerve. Of these thirty-one, thirteen became blind in three months; five, in six; nine, in twelve; and four in twenty-four.

Blindness often occurs more speedily. Indeed, among neurological traditions is one of a tabetic on board a ship in the tropics who with his fellow passengers was timing the sunset: while the sun was still on the horizon he exclaimed, "It's gone." Blindness had suddenly struck him. An engineer, aged thirty-six, went to bed, in apparently normal health, and in the darkness of a winter morning, thinking it was his usual time to rise,

struck a match to enable him to see his watch. To his astonishment, although no flame was visible, he burnt his fingers. He came to the National Hospital, London, where I was, at the time, House Physician. He seemed quite blind: he had bilateral optic atrophy: there was a probable decrease in his knee-jerks and he had incontrovertible loss of sensibility to pain (pinprick) over root areas in the lower limbs. Sudden blindness is a highly improbable consequence of mere structural change in the optic nerves, and resembles more a paretic than a tabetic phenomenon.

Syphilitic degeneration of the optic nerve fibers is irreparable. The visual loss once established is, as a rule, permanent. But Gowers and others have recorded cases of improvement of measurable extent. The partial destruction of some of many paths conveying impulses concerned in the same function, at first, especially if the destruction occur rapidly, may derange the whole function. Soon this excessive impairment passes off and the true degree of disturbance of vision alone remains. One of my patients, whose blindness developed rapidly until the visual threshold for light perception was higher than a thirty-two candle power light, at a distance of six feet, without treatment, subsequently perceived correctly nearly every change in illumination with a four candle power lamp. Moreover, with the onset of loss of vision, such profound depression and mental perturbation result that the confessed is seldom a true index to the real visual acuity. Spontaneous improvement in vision may be due to three factors; first, to the absorption of an accompanying meningeal or interstitial exudate which by pressure on the optic nerves is impeding their func-

tion; second, to the readjustment of the visual faculty to the structural defect; and third, to the recovery from unfavorable mental states. Much of the alleged remedial effect of mercury and of salvarsan is attributable to the last two of these factors. I have never been able, by the methods of examination I have described, to detect any benefit from the administration of these substances.

In 1881, Moriz Benedikt of Vienna asserted that tabes associated with rapid optic atrophy shows benign spinal symptoms.¹

He grew in optimism. In 1887,² in an open letter to Babinski in the *Wiener medicinische Wochenschrift*, he emphasized his insinuation of 1881. He proclaimed a law to which till then he knew *no exception*, that the specific motor symptoms of tabes, no matter to what degree they had attained, would diminish in severity im-

¹“Obwohl wir nicht im Stande sind auf dem Verlauf der Sehnervenatrophie irgend einen wesentlichen Einfluss zu nehmen dieselbe vielmehr gewöhnlich rapid usque ad ultimum fortschreitet so können wir doch im Bezug auf die spinalen Symptomen wenn sie noch so ausgebildet und intensiv sind die Prognose höchst günstig stellen. Eine Ausnahme machen vielleicht nur sehr veraltete Fälle. Es kann geschehen dass mit Ausnahme des Schwankens beim Stehen mit geschlossenen Augen und der Sehnen reflexe die nicht mehr zum Vorscheine kommen alle ataktischen Symptomen schwinden.”

²“Ueber die Prognose und Therapie der Tabes.” Offener Brief an Herrn Dr. T. Babinski in Paris, *Wiener med. Wochenschrift*, Aug. 14, 1887, p. 1130.

“Es ist ein Gesetz von dem ich persönlich bis jetzt absolut keine Ausnahme kenne, dass die specifischen, tabetischen motorischen Erscheinungen wenn sie einen noch so hohen Grad erreicht haben, zurück gehen, sobald die Krankheit mit Sehnervenatrophie eingesetzt hat.”

“Ich habe seit dem z. B. einen Fall (Case 399) mit prodromaler Sehnervenatrophie beobachtet der absolut nicht mehr im Stande war zu stehen und einen Schritt zu gehen, der aber unter entsprechender Behandlung so hergestellt wurde das er jahrelang obwohl total erblindet die complicirtesten Gänge in der Stadt machte.”

mediately the disease became complicated by atrophy of the optic nerve.

Déjerine and Martin,¹ in 1889, from studying one hundred cases of tabes, eighteen of which showed optic atrophy, concluded that optic atrophy occurring at the beginning of tabes arrests almost always the development of sclerosis of the posterior columns, and at the same time diminishes the sensory symptoms. So far, they state, "*la proposition de Benedikt est exacte.*" But they believed they had demonstrated that once the incoordination is established, blindness does not influence the further course of the disease.² Martin, in his thesis published the following year, dealt with twenty-one cases of tabetic optic atrophy and reiterated the views he had already expressed with his master. Raymond tends to agree with Déjerine and Martin. Ingelrans in 1897 supported Benedikt's "law" in its entirety. So did Förster in 1900; and to a greater or less extent so also did Benenati in 1901, and Schüpfer in 1901.

Buzzard, Léri and others have opposed the law.

Ataxia is an inaccurate guide to the extent of the spinal lesions, yet it has been used by those who opposed and those who upheld the idea that optic tabes benignly influences spinal tabes. The cases published in this discussion show ataxia may follow optic atrophy; and optic atrophy may follow ataxia. Optic tabes, therefore, does not preclude spinal tabes nor does spinal tabes preclude optic. They further show that ataxia usually does not

¹ "*L'atrophie papillaire survenant au début du tabes arrête presque toujours l'évolution de la sclérose des cordons postérieurs en même temps qu'elle diminue les symptômes d'ordre sensitifs.*"

² "*Lorsque l'incoordination est établie la cécité n'influe en rien sur la marche de cette dernière.*"

follow optic atrophy nor does optic atrophy usually follow ataxia; and that tabetics with optic atrophy have more seldom spinal lesions than have those without optic atrophy.

There is in this evidence no proof of any arrest of optic atrophy by spinal tabes nor of spinal tabes by optic atrophy; but there is proof that the chief stress of optic tabes falls upon the optic nerve and not upon the spinal mechanism; and that the chief stress of spinal tabes falls upon the spinal mechanism and not upon the optic. There is also proof that tabes beginning in the optic nerve is often limited mainly to it.

If there is a causal relation between optic atrophy or blindness, and the retention of the power of coördination, then this relationship should be most evident when the optic atrophy reduces vision to a degree which precludes all the aid to movement normally derived from sight.

Indisputably, blind tabetics are less prone to ataxia than seeing tabetics. From comparing blind with seeing tabetics, we can reach no conclusion as to the existence of any influence of blindness upon ataxia, unless the contrasted blind and seeing possess equally extensive incoördinating lesions. To demonstrate the precise extent of the incoördinating lesions is difficult. If series of blind and seeing tabetics with equally extensive lesions were collected and examined, the ataxia might be found to vary among the seeing just as much as between the average seeing and the average blind ataxic. The only conclusive evidence of the influence of blindness upon ataxia is obtainable by clinical observation of the effect of blindness supervening upon ataxia. When in ataxic cases blindness supervenes, no question of the extent to which the tabetic lesions affect the cord can arise, for

before the onset of the blindness the lesions were such that ataxia had already arisen. The cord lesions were present, the ataxia existed, blindness supervened, what was the result?

Benedikt, in 1887, stated, and I cannot find that he subsequently modified his statements, that he had never yet seen a case in which improvement in spinal symptoms did not occur. His statement implied that he had seen cases in which improvement did occur. I have searched his publications and found only one case. He makes such use of this single case that if he had had others, he would have been likely to disclose them. Martin (1890) reports one case of marked improvement in ataxia when complete optic atrophy occurred, but he remarks that remissions in tabes are frequent, and that this isolated example does not suffice to establish a causal relationship between the blindness and the disappearance of the ataxia. He was dealing with a blind tabetic; any example, however isolated, of such a strange phenomenon as permanent remission of ataxia associated with blindness, demands explanation.

Bouchaud records a case in which, between 1875 and 1882, there were among other symptoms much motor trouble, disordered movements of the lower limbs, and some incoördination of the upper limbs. In 1882, the vision began to fail: by 1884, blindness was absolute, but the symptom, ataxia, had ameliorated: in 1891, the incoördination had disappeared: not even Romberg's sign remained.

Inglérans records two tabetic women in Déjerine's service who were definitely ataxic and whose ataxia disappeared when they became blind. Madame A. (Observation xxix) became ataxic in 1882, and blind in 1884;

her ataxia subsequently disappeared; in 1897 she still walked correctly. Madame Del. (Observation xxxvi) was ataxic from 1885, and developed blindness in 1889; soon after, the ataxia began to disappear; in 1897, she had no signs of motor incoördination.

Förster describes two cases of ataxia (cases 15 and 19) in which so great improvement followed the onset of blindness that the disease returned to the preataxic stage. In one, incoördination was evident only in running; in the other, only in the dark.

Benenati (1901) reports two cases of ataxia ameliorated by the onset of optic atrophy. The first, a man, had normal vision in the right eye. The improvement in the ataxia Benenati attributes to the complete optic atrophy in the left eye. This case certainly should not be held to prove anything. Loss of one eye in normal life does not influence equilibrium. The other case, a woman, had gray atrophy, but all the information given is, "*La vista è molto ridotta in ambo gli occhi.*"

Schüpfer (1901) reports five cases of improvement in ataxia following the onset of optic atrophy (cases 2, 5, 11, 23, 24). Case 5 he disallows because he thinks the disturbance of locomotion was due to the onset of blindness. But when these disturbances appeared, the patient had still one-tenth vision in both eyes. One-tenth vision enables form to be distinguished and, therefore, is not an adequate explanation of "*disturbi dell'equilibrio*" (p. 253). Three years later, "*deambulazione ottima, assenza di atassi*" (p. 254) is reported.

I am indebted to Dr. Isador Abrahamson, neurologist to the Montefiore Home, New York City, for the following interesting example: Max Krasnow, in 1906, at the age of 40, was attacked with lightning pains followed

by numbness of the right leg. Six months later vision began to fail, first in the left eye, then in the right. In the year 1906-07, ataxia appeared. As vision deteriorated, the ataxia increased. About the beginning of 1908 he became totally blind. He entered the Montefiore Home on April 24, 1908. His gait then was wildly ataxic. He began spontaneously to walk better and continued to improve till his ataxia disappeared. He now is so steady that he acts as a prop and a guide for other ataxic patients (Fig. 42).

Léri reluctantly admits one case (Harr, p. 115). Harr was very

ataxic from 1888-1890; in 1889, while under observation in Bicêtre, his blindness developed: according to Léri, Harr now (1903) walks more as a blind man than as an ataxic.

I have demonstrated three cases, one of which is now in the Neurological Hospital, to the Students at Fordham University and at the New York Post-Graduate Medical



FIG. 42.—Dr. Abrahamson's case. Max Krasnow (on the reader's right) while able to see was very ataxic. Since he became blind his ataxia has disappeared and his attitude has become normal again. He is here shown aiding a seeing ataxic to walk.

School. But only in one of them, Rose, did improvement occur under my observation. The others gave a history of ataxia, but could all stand and walk coördinately when I first saw them. The ataxia in Rose was slight; he now shows no trace either of ataxia or of swaying.

Léri objects to accepting the word of the patient regarding the degree of ataxia before and after blindness. The patient is the best judge of his own walking ability which he tends so little to exaggerate that Léri's objection to this evidence seems scarcely justified. If all cases in which the improvement in ataxia did not occur under medical supervision be excluded, the exclusion affects Martin's case, and one at least of Schüpfer's, and the two I have demonstrated. Both of Benenati's should be eliminated, because in neither did blindness exist.

Cases have been reported of ataxia disappearing after the onset of blindness in which some remedial measure, such as hot baths, galvanism, etc., was alleged to have induced the improvement. These, which I have not quoted, include Benedikt's solitary case, for that patient at some time subsequent to his blindness received much electrical treatment, to which Benedikt attributes some of the credit for the lack of ataxia.

There now remain as indubitable cases in which improvement of ataxia occurred spontaneously after the onset of blindness, Bouchaud's case, Léri's case, Abrahamson's case, two of Inglerans', two of Förster's, three of Schüpfer's, and one of mine, a total of eleven.

The claim that ataxia may spontaneously decrease in blindness rests, so far as I know, only upon these eleven cases. One incontestable case would suffice to prove the fact. But the cumulative effect of eleven incontrovertible cases has a corroborative value far exceeding that which

estimate by arithmetical or even geometrical progression would confer. These eleven cases establish absolutely that in blindness coördination may spontaneously increase, ataxia may spontaneously decrease. Blindness, we have seen, affects not more than eight per cent of tabetics, probably less; and blindness follows ataxia in about six per cent. If blindness follows ataxia in six per cent of the syphilitic blind, and not more than eight per cent of tabetics are blind, the possibility of observing the effect of blindness upon ataxia occurs in not more than 0.48 per cent of tabetics. But Mott claims that 50 per cent of optic atrophies subsequently develop general paresis; so the opportunity to observe improvement may occur in less than 0.24 per cent. These figures do not pretend to any absolute value; they are stated only to show that the scarcity of the evidence is not due to the exceptional nature of the fact which it establishes but to the rarity of cases which yield incontrovertible proof of that fact.

Byrom Bramwell reports a case of a tabetic in whom, after two years of blindness, ataxia appeared, and advanced rapidly till the patient could not stand; among others, both Martin and Léri report two analogous cases.

Simultaneously with the onset of blindness ataxia may develop. Such cases have been observed by Pearce Bailey, Schüpfer, myself and others. Or if the ataxia is already established, as in Abrahamson's case, it usually first becomes aggravated by blindness.

The benign influence of blindness upon ataxia is, therefore, neither invariable nor inevitable, nor is it immediate, but develops some time after vision is lost and tends to increase with the duration of the blindness.

As long as an ataxic sees, he is a creature at the mercy of his environment. By day his environment is changing constantly; he is ever striving to keep pace with it; ever painfully alert and watchful; confronted with a fresh problem wherever his gaze rests. He must constantly fight against his economic disability. He is harassed, perplexed and fearful. He doubts and dreads every influence he does not see.

The blind tabetic lives a life of enforced inaction; he is outside all social strife; he reacts only to his immediate surroundings, to the simple reflex problems which penetrate his darkened life. He sees less; therefore, fears less, worries less, thinks less. He may be depressed but he is less fatigued and less fearful than the seeing tabetic. His mental state, therefore, improves; his capacity for effort and for attention increases. His postural sense claims its share in the attention bequeathed by vision. The longer the blindness lasts, other things being equal, the more the mental state improves. With this improvement the psychic influences unfavorable to coördinate movement diminish: and in the constant calm in which the blind tabetic exists, necessity and lack of distraction confer an interest upon the hitherto despised and suppressed postural images. They still remain feeble, few, and imperfect, but he learns to appreciate them, to interpret them, to discriminate changes among them. As he gains knowledge, he gains confidence, he gains power to move.

The law of the relation of loss of vision to ataxia, if it can be dignified as a law, should be: *Ceteris paribus*, of two tabetics, at any stage of the disease, one blind, the other seeing, the blind has the greater tendency to persist coördinate, the lesser tendency to become ataxic.

The degree of this compensatory improvement in the power of coördination will, *ceteris paribus*, tend to rise with the duration of the blindness to a degree depending upon the training capacity of the tabetic. Slinger and Horsley observed that the postural sense of the non-tabetic blind ending a course of training was one-fourth greater than that of those beginning their training (*see* p. 59). The amount of improvement in movement which may occur spontaneously in blindness is usually remarkable; and may be such that no discernible sign of incoördination can be detected.

In Bouchaud's case the ataxia began to decrease soon after the blindness occurred in 1884, and in 1891 not even Romberg's sign was present. Inglerans' observations show a similar improvement: the whilom ataxic, Madame A., still walked correctly after fifteen years of blindness, and Madame Del., after nine years, showed no sign of reverting to incoördination. Léri's case, Harr, did not lose his ataxia till he had been one year blind; after fourteen years he continued to walk more as a blind man than as an ataxic. Abrahamson's case, after a temporary aggravation of the ataxia, showed gradual improvement, till at the end of six years there was no sign of ataxia.

As blind tabetics are less ataxic because they are blind, the long preataxic stage of the blind is not solely an accident of localization of the tabetic lesion in the optic nerve, but is due in part to the tranquil mental state which blindness ensures. In other words, the blind tabetic tends to remain preataxic after his spinal lesions have passed a point at which a seeing tabetic would have become ataxic. Déjerine, Babinski, and others have

found in autopsies upon blind tabetics with no ataxia, astonishingly widespread spinal lesions.

The relations of blindness to ataxia may be postulated as follows:—

I. Improvement in ataxia will result from blindness after an adequate interval has elapsed:

(a) When incoördinating lesions remain stationary, and the mental capacity for training is not destroyed by cerebral disease.

(b) Even if the structural tabetic lesions leading to incoördination extend, provided that the effect of this extension be less than the compensating influence of the blindness upon the mental state.

II. No change in the ataxia will follow loss of vision if the advance of the incoördinating lesion just neutralizes the beneficial influence of the blindness. Obviously this stationary phase can be but temporary.

III. Increase of ataxia in spite of the blindness arises:

(a) When the incoördinating lesions advance faster than the coördinating tendency of the blindness grows. Here increase in other morbid signs (spread of sensory and of visceral affections) will show the progress of the ataxia to be merely one expression of a general advance in the spinal lesions and will serve to distinguish it from

(b) When blindness is accompanied by mental deterioration. Mott's statement that fifty per cent of early optic atrophies develop general paralysis is interesting as affording a possible explanation of cases of this class.

Less evident but no less certain is the good effect exerted upon pain (Inglersans, Déjerine) and deafness (Benenati) by the improvement in the mental state which follows blindness.

Diplopia is said to occur in about forty per cent of tabetics. With the exception of lightning pains it is



FIG. 43.—Paralysis of the left third nerve. The tabetic is here seen trying to look to the right. The left eye rotated outwards but the right remained immobile.

often the earliest symptom which tabetics notice. It may last only a few seconds, minutes, hours, or days; sometimes even weeks, but seldom longer. It may recur unexpectedly. If of short duration, it may be forgotten by the patient unless recalled to his memory by questioning. The degree of disturbance varies from blurring of an object to its duplication. Rarely more than two images are seen.

Sherrington has shown that the visual images evoked

by impulses from one retina, during their elaboration, remain distinct from the visual images which are evoked simultaneously by impulses from the other retina. The resultant of the images from the one retina then fuse with that from the other to produce the single sensation



FIG. 44.—Complete paralysis of the fourth nerve and partial paralysis of the third. The patient could not move her eyes upwards. To look up she tilted back her head.

of binocular vision. When the binocular mechanism contributes two resultants not such as we normally fuse, we lose binocular vision; two distinct sensations arise, two conscious images of one object.

After a more or less unsuccessful attempt to fuse the two dissociated images, the tabetic ignores the false image; suppresses all its associations; and, at the cost

of the binocular quality, recovers his visual accuracy. Hence, the diplopia is transient.

When one optic nerve is partially destroyed, stimulation of that retina may result in few, feeble and delayed impulses passing the lesion. These impulses evoke visual images which are less perfect and less intense than those from the sound eye. Besides thus differing in nature, the images from the two eyes are asynchronous; hence they may not fuse. When the site stimulated in one retina does not bear the usual correspondence to that stimulated in the other, visual images which are not complementary are evoked and do not fuse. Hence, diplopia develops when the accommodation mechanism is disturbed; or when, owing to vestibular lesions, the coördination of the eye movements fails; or when, through implication of the sensory or motor innervation of the eye muscles, the eye movements are not correlated. Examination of the optic nerve, of the accommodation apparatus, of the vestibular nerve and of the eye movements, will disclose the underlying factor in diplopia. The diplopia which results from weakness of one or more of the external muscles of the eye, consequent upon syphilitic lesions in the nerve supply, is accompanied by abnormal position and limitation of movement of the eye. If the patient comes under observation while suffering from diplopia, the candle test will reveal the faulty muscle. Usually, however, the investigated tabetic has already learnt to suppress the false image. Even with evident strabismus there may be no diplopia. Mirallié and Desclaux recommend for the detection of the suppressed image the use of a conical paper tube, 25 cm. long, 4 cm. in diameter at the base and 1 cm. in diameter at the apex; and of a dark colored circular patch, 3 cm.

in diameter, in the middle of a large white card. When the card is 5 or 6 meters distant, if the patient who has a suppressed image places the base of the cone to one eye and looks at the dark central patch, he sees, not one,



FIG. 45.—Complete paralysis of the right sixth nerve. This tabetic is trying to look to the right. She instinctively turns her head to the right, her left eye rotates imperfectly to the right (partial left third palsy) and her right eye, instead of moving outward, remains gazing directly forward.

but two, patches. The amount of the separation of the true and the false patches and their relative position vary with the cause of the diplopia.

Instead of the large white card, I used a white screen in which concentric circles of regularly graded diameter enclose the central colored patch, and radii pass from the colored patch to the circumference of the outermost

circle. These circles and radii enable the position of the false image to be easily defined.

Of twenty tabetics, without refractory errors and without conscious diplopia, seventeen were induced by this



FIG. 46.—Complete right and partial left ptosis in tabes.

means to see double; five saw double when the tube was placed at either eye; twelve with the tube at one eye, not at the other; nine of the seventeen gave a history of diplopia.

The internal rectus was at fault eight times; the superior oblique thrice; the external rectus twice; in the remaining four I could not with certainty determine the erring muscle.

The diplopia tends spontaneously to disappear. So also in some instances does the strabismus. Strabismus is, as are all other motor disabilities of tabes, very amenable to psychotherapy. Drooping (ptosis) and retraction of the eyelids also occur in tabes.

Complete lesions of the fifth nerve, the common sensory nerve of the face and of the buccal, nasal, ocular and, in part, of the aural mucous membrane, or of its sensory roots, are rare. So, also, are lesions of the motor root which supplies the muscles of mastication. But partial and bilateral sensory disturbances in the face are more common than is generally supposed. There is nothing noteworthy in the blunting of sensibility detectable here except for one fact: the few I have examined did not show the root character observable in sensory loss in other regions.

Absence of nausea from pharyngeal irritation, absence of pain and of lid closure when the cornea is touched are often present in tabes. The sensory paths of these reflexes are not yet surely identified.

Rarely facial palsy occurs. It may appear very early in tabes and is usually unilateral. Fournier mentions eight cases among two hundred and twenty-three pre-ataxic tabetics. Unilateral disturbances of muscles which are bilateral and which habitually function together, tend rapidly and spontaneously to disappear. The tabetic facial palsy is characteristically transient, vanishing in a few days or weeks. Some of the cases described are merely results of banal meningeal inflammation. The site and the nature of the condition do not warrant fuller discussion here, but I hope later to publish the data I have collected concerning them.

The eighth nerve has two parts, a vestibular, and an

auditory or cochlear. Both may be attacked by the spirochete. The source, connections and functions of the vestibular nerve have already been described (pp. 29 and 30). Vestibular lesions are more frequent than auditory. They may occur early and be unilateral or bilateral. Lesions of the vestibular nerve produce loss of muscular tone and loss of the sense of the body's posture. Hence dizziness, swaying, diplopia, nausea and other symptoms arise. Lesions of the vestibular nerve are detected by three tests: *a*, the water test; *b*, the galvanic test; *c*, the rotatory test.

(*a*) The Water Test: The head is maintained upright and the ear is irrigated for twenty seconds with cool or warm water.

(*b*) The Galvanic Test: In the external meatus or on the tragus of the ear an electrode is placed; the other electrode may be applied to the back or arm or any other site except the ear. According as the anode or cathode be placed to the ear, the result differs.

(*c*) The Rotary Test: The examined person is placed in a revolving chair and rotated about ten times in twenty seconds.

After any one of these procedures nystagmus normally appears and persists for a few seconds. From the presence or absence, the nature and the direction of this induced nystagmus the integrity or disturbance of the vestibular function is determined.¹

Wittmaack (1907) induced prolonged auditory fatigue in groups of guinea pigs. Each group was exposed to

¹For the detailed interpretation of the varieties of induced nystagmus in vestibular lesions see Barker's "Monographic Medicine," Vol. IV, Appleton, New York.

a different tone. The group tone was sounded in the ear of the guinea pigs frequently and periodically for many months. The guinea pigs were then killed, and their cochlear membranes and nerves were examined histologically. He found that prolonged overstimulation had caused the cochlear nerve fibers to degenerate. The bundle which degenerated varied with the tone; thus, for a tone of high pitch, a certain bundle degenerated; for a tone of low frequency, another bundle degenerated. This degeneration began in the sensory membrane, and spread along the cochlear nerve toward its nucleus in the medulla.

Habermann examined, by histological methods, the sensory membrane and cochlear nerve in cases of defective hearing among workers in noisy trades. He was able to corroborate Wittmaack's results.

Hence, when compound aerial waves of sound are converted into nerve impulses at the cochlear membrane, they are resolved into elemental forms, each equivalent to a component tone, and each transmitted along a separate fiber or bundle of fibers of the cochlear nerve to the medulla. Thence, they pass to the cortex to excite, in ordered sequence, images of tone.

In the brain the elemental tones which result from the disintegration of a sound at the cochlear membrane, are grouped and regrouped until the final synthesis is complete. Then we hear. Only a musician analyzes what he hears; even he distinguishes tones seldom and consciously. Others perceive merely the end result of a complex mental process.

A healthy person may not perceive sound waves which impinge upon his cochlear membrane. We who live and think in noisy cities do so mainly owing to this power

to turn a deaf ear. We experience daily in ourselves, therefore, a deafness due to distraction, preoccupation, lack of attention.

In all hearing there are, thus, two factors, a psychic and a peripheral: we hear among the sounds wherein we live only those which we wish to hear, only those to which we devote attention.

At a given moment we may not hear, owing to the competition for our attention. But patients who have a morbidly restricted power of attention may not be able to hear even when they are listening: and their deafness is remarkable in that it alters with the amount of attention available at the moment for auditory impressions. This psychic deafness exists normally in the preoccupied and in the old; and pathologically in the hysterical; and sometimes in the tabetic.

In tabes, one or both auditory nerves may be implicated. The sound of a watch ticking may be heard at a greater distance from one ear than from the other: it may be imperceptible even when the watch is in contact with the ear. The vibration of a tuning fork is heard better when the base of the fork is in contact with the mastoid bone than when the vibrating prongs are held near the ear; bone conduction of sound is better than air conduction: therefore, the disturbance of hearing is due to a nerve lesion. When the tuning fork is placed on the top of the head in the mesial anteroposterior plane, the sound is heard less in the more affected ear. According to the nature of the fibers destroyed, perception of tuning forks of various pitch is lost. With destruction of certain fibers, tones of high pitch become inaudible; with destruction of others, tones of low

pitch are lost. Together with this loss, subjective noises may occur.

In all tabetic lesions of the auditory nerve, except its extremely rare complete destruction, some power of hearing must persist; for the persistence of nerve fibers means persistence of the ability to detect the tones which these fibers subserve. So few fibers may escape that their survival is undiscoverable by ordinary routine methods of examination and can be detected only by carefully testing the apparently deaf tabetic's hearing, throughout the range of the auditory scale of the human ear, by means of the Edelmann-Bezold apparatus.

In the auditory nerve, as elsewhere, the tabetic lesions cause fewer, feebler and imperfect sensory images. Unless he particularly focusses his attention, the afflicted tabetic detects only a sound or a word here and there. To be continually on guard, to strain every nerve for chance sounds may soon prove beyond his strength. The occasional meaningless sounds which are heard are ignored, for it is speech, not isolated words, which command attention. Few auditory stimuli are, however, greater to a person than the sound of his own name, or sounds which connote things intimately related to his personality. Hence the last to be neglected are, as a rule, the personal sounds: they can be lightly heard by the tabetic even when impersonal topics must be communicated by shouting.

Sound must be endowed with this inherent interest or with a considerable degree of loudness, or with a certain imperative quality, before the tabetic may become conscious of it. Below this threshold he is deaf. If the hearing retained be not wholly inadequate for the rougher needs of life, the tabetic may become more or

less resigned to his deafness. If hearing be too impaired to permit a satisfying amount of communication with the world of sounds, and especially if subjective sounds occur, the consequences vary according to the mental state of the tabetic. The uncertainty and effort which accompany his striving to hear fatigue and depress him; and hearing shares in the depreciation which all cerebral functions suffer in such unfavorable mental states. Even if the lesion be stationary the deafness tends to increase.

Deafness is less in interest than in apathy and in favorable than in unfavorable mental states. Psychotherapy can often procure considerable improvement in hearing. Deafness depends not only on the tabetic lesions in the auditory nerve, but also upon the mental state. The amount of deafness is an inaccurate guide to the extent of the destruction of the auditory nerve, a misleading index to the actual capacity to hear.

The ninth or glossopharyngeal nerve is occasionally implicated for loss of taste may occur.

There is nothing to distinguish syphilitic lesions of the eleventh or spinal accessory nerve, the nerve of supply of the trapezius; or lesions of the twelfth, the hypoglossal nerve, the nerve of supply of the tongue, from analogous lesions of motor nerves elsewhere.

Invasion of the vagus is very frequent. With the sympathetic the vagus constitutes the vegetative nervous system. Its implication in tabes will be considered in the next two chapters.

CHAPTER VI

RESULTS OF THE SYPHILITIC LESIONS OF THE VEGETATIVE NERVOUS SYSTEM

a. SENSORY AND MOTOR DISTURBANCES

The correlation of cells. Development of the primitive vegetative nervous system. The vegetative nervous system in man composed of two antagonistic components, the vagal and the sympathetic. Action of these components illustrated by the pupil. The pupil in tabes. Vegetative reflex arcs and plexuses. The afferent and efferent fibers linking these arcs with so-called "centers" in the central nervous system. Tracing of the sensory fiber. Proof that pain from pressure on the face passes not by the fifth but by the sympathetic. Pressure pain loss in tabes on limbs, eyes, testicles and other viscera. Loss of visceral reflexes. Lightning pains, character and frequency. Their source in irritation of the vegetative nervous system. Lightning pains and crises allied phenomena. Gastric, rectal, bladder and genital crises. Pains and crises reciprocally related to the mental state. The physiology of micturition. Micturition in tabes.

THE activity of the individual cell is governed by its nucleus. In aggregations, the cells specialize; some procure, others digest, others convey food necessary to the existence of the colony. The more highly differentiated the cells are, the less do they depend for their needs upon their individual efforts, the more is the activity of the various cells correlated to the needs of the whole colony.

This correlation is earliest attained by chemical and mechanical action. Nutritive fluids are circulated through the colony by cilia and by contractile tubes; the various cells withdraw what they need, and excrete into the circulating fluid substances which stimulate the activ-

ity of their fellows. Then nerve cells are differentiated to preside over these vital activities. They send fibers among the other cells. At the ends of these nerve fibers, chemical and mechanical action is converted into nerve energy which passes along fibers to the body of the nerve cell. From this presiding nerve cell, impulses pass by other fibers to be distributed throughout the organism

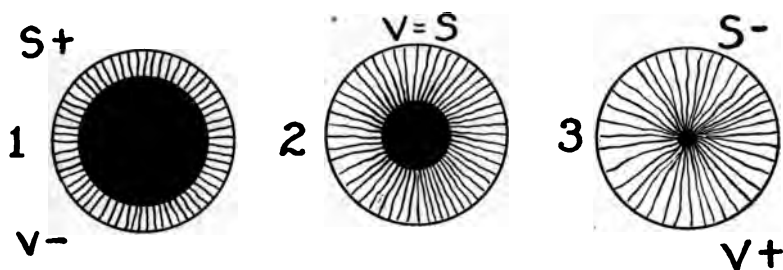


FIG. 47.—Diagrams to show the effect on the pupil: 1, of stimulation of the sympathetic ($S+$) or paralysis of the vagal ($V-$) fibers; 2, of the balanced ($V = S$) action of sympathetic and of vagal stimulation; and 3, of paralysis of the sympathetic ($S-$) or of stimulation of the vagal ($V+$) fibers.

to provoke chemical and mechanical changes. These presiding or ganglion cells and their fibers constitute the primitive vegetative nervous system through which the breaking down or building up of the protoplasm in the multicellular organism is correlated more speedily, more powerfully, and more accurately than by direct mechanical or chemical action.

In man, the vegetative system is composed of cells which during embryonic life wander from the ventral part of the neural canal towards the periphery.

Among the vegetative fibers, two antagonistic groups are said to be distinguishable in practically every organ; one, a stimulating or accelerator group, the autonomic or vagal group; the other, an inhibitory, the sympathetic group. Thus, in the pupil, stimulation of the vagal

fibers causes contraction of the sphincter of the iris and produces a small pupil (Fig. 47). Stimulation of the sympathetic fibers causes contraction of the dilatator of the iris and produces enlargement of the pupil (Fig. 47).



FIG. 48.—Retraction of the lids, eccentric pupils. (Montefiore Hospital case.)

A small pupil may result either from stimulation of the vagal fibers or from paralysis of the dilating fibers of the sympathetic; a large pupil, from paralysis of the vagal fibers

(for example by atropin or belladonna) or by stimulation of the sympathetic. Usually these antagonistic tendencies more or less balance one another (Fig. 47). Variations in the size of the pupil express the oscillations in this balance.

The pupil changes which occur in tabes are of three kinds: changes in shape, in size, and in reflex activity. The normally circular pupil may become irregular, usually first during action, later when at rest also. The irregular pupil from vagal irritation becomes usually smaller (3, Fig. 47), occasionally



FIG. 49.—Tabetic with left pupil widely, right moderately dilated.

from sympathetic irritation, larger (1, Fig. 47), than its fellow. This irregular pupil of altered size more and more slowly and feebly contracts when illumination increases, or dilates when darkness occurs. The pupil ultimately becomes fixed, immobile, during changes in light, but it still narrows during near, and dilates during far, seeing. Such a pupil, inactive to light, responsive to accommodation was first

described by Argyll-Robertson of Edinburgh, and is after him named the Argyll-Robertson pupil. It is practically pathognomonic of syphilis of the nervous system, of which it usually is the first sign. The course of this light reflex is still in doubt.

The typical tabetic pupil is a pin-point pupil, the result of unbalanced action of the vagal fibers. When these vagal fibers are destroyed the pupil may dilate, owing to the unopposed action of the sympathetic.

The cells of the vegetative nervous system are linked by their processes to form reflex arcs. These vegetative reflex arcs are linked in plexuses which are associated with a view to their collaboration in functions common to them. Thus in the intestinal wall are the associated plexuses of Meissner and of Auerbach. Through their correlated activity, while one portion of the muscular tube of the intestine contracts, the succeeding portion is caused to dilate; so the passage onwards of the intestinal contents is facilitated. As the threshold of each reflex arc which forms a link in this plexus chain is lowered in sequence, the waves of contraction pass along the muscular tube of the intestine in the same direction, in regular order, and at a uniform rate. This reflex correlation is wholly peripheral: it persists after all connection with the central nervous system has been severed.

From the plexuses, fibers conveying afferent impulses to the central nervous system pass along posterior nerve roots and homologous paths, to where in the central nervous system cell groups occur, sometimes called centers, from which small white medullated fibers arise. These small white fibers leave the pons, medulla and spinal cord by the anterior nerve roots, or by their cranial representatives, to proceed to the periphery, along blood vessels

or nerves, till they form a synapse with cells in a near or remote ganglion of the vegetative nervous system. From such a synaptic cell a non-medullated or gray fiber conveys the spinal impulses to their destination in liver



FIG. 50.—Extirpation of Gasserian ganglion. The resulting area of anesthesia to light touch is shown by the continuous line and to 65 gm./dm.² pressure, by the dotted line. Note, that although there is complete loss of common sensibility within the area of the distribution of the fifth nerve, sensibility to moderate pressure touch is lost only on the forehead and around the eye. (Maloney and Kennedy.)

or other gland, in intestine or other involuntary muscle. Chemical and mechanical stimuli, affecting the terminations of the vegetative fibers in the viscera, are converted into nerve impulses which may, in part, pass back to the central nervous system; and in cells there evoke impulses which, returning to the stimulated site or to allied sites, accelerate or inhibit the activity of the peripheral reflex arcs of the vegetative nervous system. Thus may

be correlated the vegetative activities of the several segments of the body.

But these usually unperceived impulses which arise from visceral stimulation, when they are of extraordinary intensity, may reach consciousness. We feel tumult-

tuous heart action, abnormal peristalsis in the intestines, and other visceral changes, as discomfort or even pain.

Some state that the fiber which conveys discomfort or pain from a viscus originates in the bipolar cell of a spinal ganglion. The truth or falseness of this is difficult to establish. The Gasserian ganglion of the fifth cranial nerve is the homologue of the posterior root ganglion. In extirpation of the Gasserian ganglion loss of touch, of pinprick pain and of sensitivity to light pressures occurs on the forehead, cheek, lips, nasal, buccal and lingual mucous membranes supplied by the fifth, but either no or only partial loss of pressure pain.

Pain can always be still elicited by

pressure, but the loss on the cheek is constantly less than that on the forehead, on the lips than on the cheek, and the tongue enjoys greater immunity than all of these sites. Sometimes the forehead alone shows loss: the cheek is never affected unless the forehead also is; nor



FIG. 51.—Extirpation of the Gasserian ganglion, to show distribution of loss of pain on pressure. Outside the affected area pain was felt when a pressure of $1\frac{1}{2}$ kilos per unit area was applied; inside, pressures varying from 3 to 8 kilos were needed to evoke pain. The pressure analgesia is greatest on the forehead and less on the chin than on the cheek. (Maloney and Kennedy.)

the tongue, if the cheek escapes. Immediately after operation, the pressure pain loss is greatest but recovery is rapid and often perfect.

As complete extirpation of the ganglion may leave the pressure pain sense normal, the fibers which conduct that sense do not have their origin in the ganglion. Any loss of pressure pain produced by removal of the Gasserian ganglion must, therefore, be due to accidental injury of some nerve mechanism whose fibers in varying number enter the field of operation.

Injuries of the seventh nerve, peripheral to the Fallopiian canal, produce atrophy of the facial muscles but no loss of common sensibility; whereas injury to the seventh in the Fallopiian canal may be associated in the skin, muscles and bones of the facial muscular apparatus with loss of pain produced by pressures up to four kilos per unit area. In the area of this loss of pressure pain, the sensibility to touch, pin-prick and light pressure is perfect; the sensibility of the fifth nerve and its branches is unimpaired.

Section of the hypoglossal in the neck produces atrophy, but no loss of pressure pain in the tongue.

Fibers which do not belong to the fifth nerve; which are common to the forehead, cheek, lips and tongue; which invariably occur in the region of the fifth nerve roots and of the Gasserian ganglion; which are there in greater proportion from the forehead than from the tongue; and which anastomose so perfectly that the isolated total destruction of the fibers from one site is practically impossible—such fibers can belong only to the vegetative nervous system. The vegetative nervous system must conduct the pressure impulses which evoke



FIG. 52.—Extirpation of the Gasserian ganglion. To show pressure analgesia on the forehead only. The numbers represent the pressures per unit area, in kilos, needed to elicit pain. (Maloney and Kennedy.)



FIG. 53.—Left facial palsy from a basal fracture. To show pressure analgesia over the affected muscles. The numbers represent the pressures per unit-area, in kilos, necessary to evoke pain. (Maloney and Kennedy.)



FIG. 54.—Right facial palsy of acute inflammatory origin. Common sensibility was everywhere unimpaired but there was some pressure analgesia over the affected muscles. The numbers represent the pressures per unit area, in kilos, needed to evoke pain. (Maloney and Kennedy.)

in consciousness the sensation of pain from pressure on the forehead, cheek, lips and tongue.

As the Gasserian ganglion neither gives origin to these sensory fibers nor acts as a relay station for them, perhaps the spinal root ganglion is equally unrelated to the corresponding vegetative fibers from the trunk, limbs and viscera.

French experimenters have recently shown that the

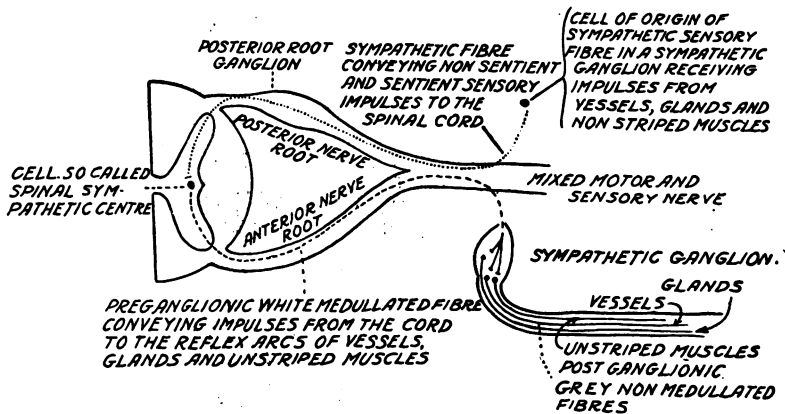


FIG. 55.—This diagram represents the relations of the spinal sympathetic fibers as analogous to those of the sympathetic fibers of the head. Thus, the sympathetic fiber conveying non-sentient and sentient sensory impulses from vessels, glands and non-striped muscle, to the spinal cord, is depicted as originating apart from the posterior root ganglion.

pain of claudication can be abolished by dissecting the sympathetic from the thickened walls of the blood vessels of the limbs.

In tabes, the vegetative fibers conducting pressure pain either because of lesions in their peripheral course or through implication in the posterior root lesion, may be partially or completely destroyed.

In the face, the normal pressure, per unit area, which elicits pain is $\frac{1}{2}$ to 1 kilo. In tabes, loss of pressure pain is occasionally met greater than that which occurs

in section of the fifth nerve or roots, after extirpation of the ganglion, and in nuclear lesions; and greater than that seen in any seventh nerve or nuclear lesion. I have examined not a few tabetics in whom pressures as high as 15 kilos per unit area, pressures at which one desists lest the tissues be injured, could be applied to the forehead, zygoma and cheeks without eliciting pain. This loss of pain on pressure in the face occurs without, or with, disturbance of common sensibility.

As a rule, loss of pressure pain appears first in the peroneal region of the lower limbs. It is there one of the early signs of tabes. This loss may at first be evident in a unilateral raising of the pressure pain threshold; on the sound limb, over the Achilles tendon, alongside the feeling of pressure, pain may develop when a pressure of about 3 kilos per unit area is applied, whereas on the affected limb the threshold may be 5, 6, or more kilos. Soon the loss may become bilateral, then equal; and often no degree of pressure will elicit pain. Indeed, ulcers over points subject to pressure may develop painlessly: and fractures of the bones may occur without even discomfort. In the arms the loss usually first appears on the ulnar side of the forearm, in the area where reflex pain of cardiac origin occurs, in the area closely associated with the region of the spinal cord from which the ciliospinal fibers arise.

The loss of pressure pain is not confined to the face, limbs or trunk. The commonest, and often the only, sign of sensory disturbance in the head is loss of pain on pressure of the eyeballs. On compressing the eye, besides absence of pain, there is also absence of the normal slowing of the heart's rate. The testicle may be compressed as much as due regard for its integrity

permits, without inducing pain. Pressure in the epigastrium may elicit no sensation. Parturition may be painless.

Preceding the loss of pain on pressure, later accompanying it, and still later disappearing, is the most constant and usually the earliest sign of tabes, spontaneous pain.

This pain may sometimes have a root distribution. Usually the patient when asked to show where his pain lies moves his indicating hand vaguely up and down his leg. Pain may appear suddenly and, after a time, either as suddenly vanish or gradually cease. Or it may be more or less continuously present and subject to unexpected exacerbation. It may occur at all times of the day: it may awake the sleeping tabetic. It may be a mild neuralgic pain which is mistaken for rheumatism, or a pain of insupportable severity.

Usually it first appears in one leg, maybe in both, less often in the back, arms, or face. As a rule, the pain shoots down the back of the thigh to the calf, terminating sometimes there, sometimes in the heel, sometimes in a toe. It may be localized to isolated areas of muscle as if fragments were being cut out or torn out. Sometimes the pain is like that from a stab or a twisting in the flesh of a sharp instrument; sometimes it is described as tearing or rending.

The sudden, excessive, diffuse and persistent nature of the pain is reminiscent of the so-called thalamic, or of the protopathic, response to peripheral painful stimuli. It is doubtless due to an irritation of the central end of diseased fibers, as it can be associated with loss of sensibility to painful stimuli. Its localization may, therefore, be hallucinatory, like the pain felt in amputated

toes; but sometimes the painful area is hypersensitive even to the slightest pressure. The fibers which conduct this pain, in part, may belong to the common sensory system, but in part also, if not mainly, belong to the vegetative system; they are probably sympathetic in origin. Sweating, pallor, or blushing, and edema of the painful area may accompany the attack, and afford further proof that the vegetative nervous system is irritated.

When bouts of pain occur not in limbs but in viscera, they are called tabetic crises. Their nature is everywhere the same; a sudden pain, diffuse or radiating in character, which attains, almost immediately, its maximal intensity, which is accompanied by vascular, secretory and motor phenomena and which suddenly, sometimes gradually, ceases and recurs.

Attacks of pain associated with vascular and secretory changes occurring in the eye region have been described by Knauer, Spiller and others. Of the similar attacks occurring at other sites, the best known is the gastric crisis. The gastric crisis consists of nausea and vomiting, preceded by stabbing pains radiating forward from the middle of the back, or by cramping pains especially in the right hypochondrium, or by discomfort or burning pain in the epigastrium, or by eructations. The pain may closely simulate that seen in gastric ulcer or in gall stone or in appendicitis, and may lead to the harmful employment of futile surgical measures. The vomiting may or may not relieve the pain or discomfort; it may start unheralded and may be almost free from nausea. X-ray photographs of the phenomenon show a diminution of the stomach shadow and antiperistaltic movements just as in vomiting from other causes. The

stomach contents are rejected sometimes with great violence: then the vomit may be mucoid or frothy. In severe attacks the vomit is colored yellow or green by bile; and sometimes it is coffee colored by blood.

The vomiting usually lasts a few minutes; recurs after an hour or more; and may be repeated five or six times for a day, sometimes for two days. Then a quiescent period of months or longer may ensue.

The crisis may be precipitated by a drug such as pilocarpin, which stimulates the vagus. During the gastric crisis symptoms of vagal irritation are evident: the pupils become pinpoint; a characteristic laryngeal stridor is often heard which enables the vomiting of gastric crises to be distinguished audibly from other forms; and intestinal colic and diarrhea may occur. The crisis is, therefore, due to a diffuse vagal irritation with its storm center in the vagal fibers which supply the stomach.

In cases, fortunately not common, no provocation of the crisis can be traced. Concomitant increase of the signs of vestibular or of vagal lesions may occasionally show the crisis to be a manifestation of an active extension of the syphilitic invasion. The process of extension soon ends. Degeneration succeeds irritation, and the crisis stops. In other cases, dietetic errors, or mental stress, aggravate a chronic irritability of the vagus which is evident in hyperacidity, eructations, nausea, and intestinal phenomena. In other cases again psychoanalysis reveals a mental incentive to nausea, which precipitates the attack. The tracing of the immediate cause of the crisis is seldom difficult.

The amount, nature, and frequency of the vomiting, and the patient's weight are the guide to the severity

of the crises. The crises are self-limiting and tend spontaneously to disappear. In some tabetics crises are frequent, in others rare, in others, again, unknown.

Gastric crises, just as pain crises, have a large mental element in their production.

With gastric crises urgency of defecation and tenesmus may occur. Sometimes these rectal crises appear alone; the tenesmus is most distressing, and bloody stools occur. The commonest type of intestinal disturbance is spastic constipation periodically alternating with diarrhea, a disturbance often seen also in non-tabetics suffering from vagal irritability. During the diarrheal attacks, or during diarrhea produced by the use of drugs, leakage of the intestinal contents may occur, often so slight that the tabetic attributes it to his carelessness and does not consider it worthy of mention.

In early tabes, irritation of the sensory nerves of the bladder may cause an imperious demand for immediate evacuation of urine, accompanied sometimes by a spasmodic retention of urine: the urgent desire to micturate sometimes cannot be satisfied, and when, by straining and by attitudinizing, urination is induced, exaggerated general shivering or pain may occur during the enforced act. Preceding and accompanying urgency of micturition is often a sudden, intense and shooting pain which radiates along the penis and into the testicle, sometimes into the perineum. While the pain lasts the difficulty of micturition may be tremendous. An agonizing effort may result only in a few drops of blood-stained urine. During such an attack I found that the temperature of the urine as it issued from the meatus was 1.2° F. higher than the mouth temperature. After this attack the tem-

perature of the urine was 0.4° F. higher than that of the mouth.

The asocial influence of these excretory disturbances is obvious; their effect upon the mental state of the patient is sometimes profound. A tabetic who was under my care in the Post-Graduate Hospital had urgency of urination and defecation, loss of one Achilles jerk, pin-point pupils, and some hypalgesia in the legs. He walked miles out of his way, to and from his work, that he might pass many places where he could seek immediate relief when the desire to evacuate assailed him. He became so emaciated and exhausted from anxiety and from the exertion of walking, that he could hardly stand. A fortnight of rest and of evacuating at frequent regular intervals restored him.

Occasionally genital crises also arise. Slight peripheral irritation produces persistent and painful erections. The frequency with which sexual excess is blamed for the onset of tabes suggests that an irritative condition of the pudic nerve may often be an early state of tabes. With the development of anesthesia, loss of satisfaction may occur. Lack of sexual power develops, partly due to interruption of the reflex arcs; mainly, however, not to physical but to psychic causes. The blind tabetic usually remains potent. Ataxics, while being successfully trained to walk, frequently volunteer that they are recovering virility. One of my ataxic patients, H., for eight years a tabetic, for three impotent, with urinary incontinence, on his recovery from ataxia, in spite of my protests, married.

Pains and crises are worse in fatigue, depression and fear; in the seeing than in the blind (Inglerans, Déjer-

ine and others); in ataxics untrained to coördinate movement than in those trained.

The pains or crises may last for years before any other symptoms are detectable. Indeed, some clinicians distinguish types of tabes in which pains and gastric crises predominate. If the disease advances, the irritation may be followed by destruction; pains and crises then diminish and may be succeeded by paralytic phenomena.

Normally the muscular wall of the bladder contracts reflexly upon its contents at regularly recurring intervals. As the bulk of the contents increases the more powerful and more frequent these reflex contractions become, until they reach an intensity at which the contraction of the sphincter closing the bladder is inhibited; the urine is then propelled along the urethra to the exterior by a tonic contraction of the bladder wall. In the act of expelling urine from the bladder, the diaphragm is fixed, and the abdominal wall contracts, so that the general intra-abdominal pressure is increased to reinforce the expulsive power of the contracting bladder wall.

During infancy the bladder fills and empties reflexly. We are trained to consciousness of the act of micturition and taught to inhibit the act in conformity with social usage. The inhibition of the bladder reflexes produces discomfort. This discomfort must arise periodically when the bladder wall contracts on its contents, yet we are not normally subject to sudden and regularly recurring demands to micturate. The intermittent sensory stimuli from the contracting distended bladder, in their passage to consciousness, are mitigated and made continuous; and they gradually increase in their power

to command attention as the bladder fills till the sensation they evoke prevails over other interests.

The call to urinate prevails at a moment which depends not only upon the bulk of the bladder contents but upon environmental conditions. Where, as in war, the satisfying of natural impulses is dangerous, the bladder will tolerate for hours urine in quantities which under safer circumstances would demand immediate evacuation.

Once urination has begun, it tends reflexly to continue till the bladder is empty. Only the adult can interrupt the act and he, it may be, not successfully.

As the bladder develops from both allantois and cloaca, its sensory nerves pass through both lumbar and sacral posterior nerve roots. If these roots are affected by a tabetic lesion, the sensory impulses become fewer, feebler, and delayed. As the sensory loss increases, the bladder must contain a greater and greater quantity of urine before the need for evacuation becomes perceptible; while the urine flows, the tabetic may experience no sensation of its passage, and during evacuation the desire to micturate may be satisfied before the bladder is emptied.

Loss of sensibility similar to that present in the bladder and the urethra may, though usually later, be found also on the perineum and in the vagina and rectum.

The anesthesia of the urinary tract removes the only incentive to urination that the adult acknowledges; it abolishes also the demand for complete emptying. The tabetic, therefore, urinates more and more seldom; and he less and less completely empties his bladder. The bladder in consequence becomes gradually more distended and the residual urine steadily increases. The chronic over-distention mechanically impairs the blood

supply of the bladder muscles, and the overstretched, ill-nourished muscles weaken. The muscles of the bladder wall may also sometimes weaken because of the involvement of the nerve fibers of their tone reflex in a tabetic lesion.

The unperceived excess of urine in the anesthetic and muscularly weak bladder may mechanically force the stretched, weakened sphincter, especially during coughing, or sneezing, or lifting, or bending, or other circumstances in which the intra-abdominal pressure is suddenly raised. A few drops of urine escape along the insensitive urethra and the patient is astonished to feel the unexpected wetness on his thighs. Often the initial, and sometimes the only, accident of this kind occurs, not during exertion, but during the sphincter relaxation of sleep. These accidents increase in frequency. The urine-soaked garments of the tabetic are an unpleasant feature of most neurological clinics.

The weakness of the expelling power renders micturition not only an incomplete but a lengthy process. The stream has not force adequately to distend the urethral walls, so it issues small, may be forked; and, instead of continuing powerfully forward, falls immediately from the urinary orifice. Indeed, there may be no stream, merely a discontinuous trickle, which the tabetic must direct away from his person.

The tabetic may lose all desire and urinate only from a sense of expediency or from the wish to evade dribbling. The act is then performed in complete isolation; he sits or kneels or bends, presses on his abdomen, coughs and evokes the aid of the sound of running water. Sometimes, he can urinate only if immersed in a bath. And when thinking the act complete, he adjusts his

garments, he may be annoyed by continued dribbling. Immediately after urination, the tabetic bladder may yield five or more ounces to a catheter.

The intermittent trickle of urine in tabes is not like the flow from a fistula; nor is it a paralytic incontinence. It is purely a mechanical effect analogous to the overflow which occurs from a bladder with an enlarged prostate. There is no leakage in tabes unless the bulk of the urine exceeds the containing power of the bladder: dribbling in tabes results solely from over-distention, the product of infrequent and imperfect emptying. If, by periodic evacuation, the bladder be prevented from over distending, the muscle gradually recovers much of its tone and the dribbling stops.

Neglect may lead to fatal cystitis.

CHAPTER VII

RESULTS OF THE SYPHILITIC LESIONS OF THE VEGETATIVE NERVOUS SYSTEM

b. DISTURBANCES OF CHEMICAL CORRELATION

Action of the products of the internal secreting glands. The antagonistic reaction to chemical substances shown by the vagal and the sympathetic components of the vegetative nervous system. Oscillations in the balance between vagal and sympathetic components, due to chemical stimuli, control metabolism, and defense against organismal invasion. Disturbance of this control. Trophic conditions: ulcers, infections, fractures, arthropathies. Defense against the spirochete. Production of defense substances. Stimulation of vegetative nervous system by defense substances. Adequate stimulation causing perfect defense; inadequate stimulation permitting persistence of the spirochete. Inadequate stimulation due to mildness of pretaabetic lesions: to glandular insufficiency: to age: to mental stress. Imperfect defense; causes. Effect of lesions of vegetative nervous system upon defense. Why optic tabes is abortive. Why the vegetative nervous system, although the first, is not, as a rule, the main site of spirochete invasion.

IN the body cellular masses occur called internal secreting or endocrinous glands. These cells produce chemical substances commonly but, as a rule, incorrectly called hormones, that are poured into the blood and conveyed by it throughout the body. Our knowledge of hormones is slight. The hormone of one endocrinous gland is said to excite the secretory activity of another, or inhibit that of a third. A hormone is supposed to reinforce or to antagonize, completely, or in part, the activity of another hormone. These hormones, by their direct action, modify the biochemical processes of all tissue cells, mainly in the course of a slow general diffu-

sion by the blood and lymph, but partly by stimulating the receptors of the vegetative nervous system.

The sympathetic and the vagal (autonomic) components of the vegetative nervous system show towards one hormone, adrenalin, and towards other chemical substances antagonistic reactions.

Adrenalin, the hormone of the suprarenal gland, stimulates the sympathetic; the resulting increase of irri-

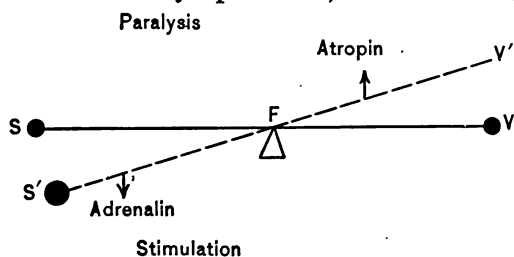


FIG. 56.—Diagram of autonomic or vagal (V, V') and sympathetic (S, S') interaction. S F V represents the balancing of vagal action by sympathetic. S' F V' shows the effect of stimulating the sympathetic by adrenalin or of paralyzing the autonomic with atropin.

tability, or tone, in the sympathetic is accompanied by diminution of tone in the vagal component, and, in consequence, by decreased activity in the pancreas and in the

thyroid glands. Cholin produces increase of tone in the vagus with concomitant diminution of tone in the sympathetic. Most of the manifestations of anaphylaxis are attributable to increased vagal irritability. The increased vagal tone may be counteracted by paralyzing the vagus with atropin or by stimulating the sympathetic with adrenalin. Oscillations in the balance of tone between these two antagonistic components of the vegetative nervous system, brought about by chemical stimuli, control metabolism, and the kindred function of defense against organismal invasion.

The cellular confederacy, which we call the human body, may be disturbed locally by the implication of the vegetative reflex arcs in the tabetic process, and generally

by the destroying of the balance of tone between the vagus and the sympathetic: then, cellular activity does not correspond to the needs of the body. Thus, pressure normally tolerated with impunity on the sole of the foot may give rise to ulcers; trophic changes may appear in the toe nails and skin; infections may readily occur around the nail bed, and the bones may become so brittle that they fracture even from the feeble strain of their weakly contracting muscles. Such fractures may be multiple. The tendency to fracture is said to be lessened by administration of the thyroid and parathyroid glands. I once saw the bones of the leg markedly atrophy. As a corollary to this rarefaction of the bones, an overgrowth¹ occasionally occurs. The bony overgrowth is painless and develops at any stage of the disease. It is often a sequel to injury. Touch may reveal, especially in cases with effusion, a higher temperature over the affected joint than over its



FIG. 57.—Trophic ulcer on the plantar aspect of the big toe of a tabetic. The prehensile attitude of these toes was habitual.

¹ Its cause has occasioned prolonged controversy. Stargardt sees in it an osteitis analogous to syphilitic inflammation elsewhere. Most believe that the proliferation is comparable with that which occurs in syringomyelia, and that it is wholly due to the nervous lesions of tabes.

unaltered fellow. When the arthropathy attacks the tarsometatarsal joints, perforating ulcers may be associated with it. Arthropathy may reinforce hypotonia in producing dislocations.

As the syphilitic infection early becomes general, the



FIG. 58.—Trophic ulcer on the outer margin of the foot of a tabetic.

order in which the tissues are attacked must be the order of their vulnerability. The lateness of nervous lesions in syphilis implies therefore that the tissues of the central nervous system possess a resistance to invasion by ordinary strains of spirochete greater than the resistance of tissues which are earlier invaded. Noguchi and Reasoner have emphasized this resistance of nerve tissues. But Noguchi (*Presse médicale*, 1913, viii, 1905) has done more. He has shown that the nervous system of rabbits which

have been frequently inoculated intravenously with spirochetes throughout a period of five months, loses this refractory tendency; that repeated intravenous infections sensitize the nervous tissues to spirochete invasion. In other words the body's reaction to the spirochete is not a local but a general biochemical response. This gen-

eral alteration in the cellular response was further demonstrated by the experiments of Finger and Landsteiner: these observers were able in human beings suffering from secondary syphilis to induce by spirochete inoculation only secondary lesions; and in others suffering from tertiary syphilis, only tertiary lesions. Krafft-Ebbing failed to produce any lesions in non-nervous tissues by



FIG. 59.—Taboparesis. Bed sores over heels, sacrum, buttocks, back and sides. (Jelliffe.)

inoculating subcutaneously a series of nine general paretics, with scrapings from primary sores.

In a person infected with syphilis, when the tissue first invaded finally heals, the spirochete next grows, not in it, but in other tissues. The cells of the first attacked tissue in repelling the invasion acquire power to resist the spirochete and acquire an immunity to the invasion greater than that which other tissues now enjoy. This has long been known from clinical observation, and has also been experimentally demonstrated. Thus, Truffi, P. Ullmann and Tomaszewski report that in dogs a second syphil-

itic infection, seven to nine weeks after a first, may be entirely without visible effect. There is, however, usually some reaction, so these observers conclude that the immunity acquired by the tissue in its defense against



FIG. 60.—Painless tabetic fracture of the head of the tibia.



FIG. 61.—Charcot left knee and ankle with trophic changes in the toes.

the first injection is, as a rule, not absolute. Perhaps the amount of reaction which the second injection evokes is partly an index, not of failure of the tissues to acquire complete immunity to the first, but of the biological difference between the spirochete of the second injection and that of the first.



A



B

FIG. 62.—Tabetic joints: A, Charcot right wrist; B, its x-ray.

Tomaszczewski and his fellow-workers showed that immunizing the eye bestowed no detectable immunity on the skin. The careful seeking of competent observers has failed to reveal the presence of defense substances.

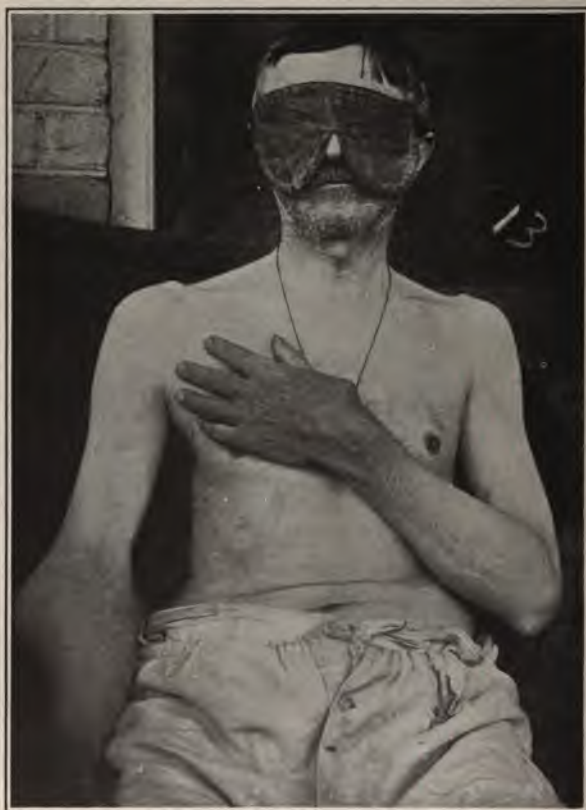


FIG. 63.—Charcot elbow. This tabetic also had wasting of the muscles of the left thumb.

Such failure may imply merely faulty methods of search. Ehrlich states that substances which activate salvarsan are present in the blood and absent from the cerebrospinal fluid. Syphilitic lesions occur in crops separated by periods of outwardly perfect quiescence; the beginning of such a period synchronizes with the healing of



Fig. 64



Fig. 65

Figs. 64 and 65.—Proliferating osteitis of lower left ribs in a tabetic. (Jelliffe.)

the lesions, with the destruction of the spirochete; the end of the period with renewed growth of the spirochete, with fresh lesions; similar but longer periods may occur between successive invasions of different types of tissues, and before the first nervous symptom appears,



FIG. 66.—Experimental brain syphilis produced in a rabbit by sensitising through repeated inoculation with the *Spirochaeta pallida*. (Noguchi.)

years may elapse. A single injection or a few crops of lesions of inconsiderable extent may immunize a whole tissue.

After a staphylococcal infection, a guinea pig immunized against many thousand minimal lethal doses of tetanus was observed by Roux to succumb to one: a dissociation of the loose innocuous chemical combination between the tetanic toxin and antitoxin occurred owing to the staphylococcal infection. Chills and bacterial infection may be followed either by the onset of tabes, or, in tabes, by marked progress in the lesions.

The quiescent periods between the lesions, the influence of time in decreasing the resistance, the immunizing of whole tissues from points of successful defense in them, and the effect of intercurrent infections, show that in syphilis defensive substances are produced. These defensive substances issue not only from invaded cells but also from cells not immediately menaced, which are stimulated to resist by products derived from attacked cells and from the spirochete. There is a concerted and correlated response of the tissue cells to the chemical substances which arise during spirochete invasion.

In the normal course of spirochete invasion, owing to successive immunization of the skin, mucous membranes and deeper tissues, a general defensive response is engendered which causes the final quiescence of the spirochete, or makes the uninvaded tissues immune. Lack of the normal chemical stimulation, due either to absence or to inadequacy of the local defense reactions in the skin, mucous membranes and deeper tissues, may prevent the proper excitation of the vegetative system and may thus prevent the perfect correlation of the chemical confederacy which is necessary to ensure de-

fense of nerve tissues against the spirochete. The occurrence of tabes after no or mild pretabetic lesions may be due in part to the insignificance of these lesions.

In the aged the activity of the endocrinous glands diminishes: Pitres has emphasized the rapid course of senile tabes. Joffroy, Oppenheim, Pierre Marie and others have noted the association of tabes with disease of the thyroid gland. Oppenheim has emphasized the value of pituitary extract in tabes. The fulminating spread of tabetic lesions which occasionally follows salvarsan injections is said to be obviated by the use of adrenalin.

The average age of onset of tabes is approximately 37 years. About this age most acquired insanities begin; carcinomata usually appear. It is the average limit of endurance, the age when the balance of mental and of vegetative life fails, owing to the stress of living.

The tabetic often volunteers the statement that his symptoms are due to prolonged worry. He may be able even to prove that they commenced soon after a shock he experienced. Tabes so frequently follows an accident that Hitzig incurred ridicule by teaching that accidents liberate toxins which lead to tabes. The physical accident to which tabes is attributed is seldom severe. Minop of Moscow showed that slight lacerations of the lymph and vascular capillaries of the cord occur after severe accidents to the spinal column, which have not destroyed the continuity of the cord. But such changes are not confined to the root zones nor to the dorsal columns. Such changes cannot produce only the signs which are discovered in cases of tabes attributed to accident. The importance of the injury, as a factor in causing tabes, lies not so much in the immediate physical

consequences as in the mental effect of the shock and the pain.

The ratio of *tabes* to syphilis is higher among Europeans than among Chinese, Persians, Egyptians, and negroes; among men than among women; and among the intellectual than among the laboring classes. In the French and Austrian armies painstaking statistical inquiries have shown that the ratio of *tabes* to syphilis is remarkably higher among the officers than among the men.

The races in which *tabes* is rare, in which the resistance to the spirochete is not imperfect, enjoy in common a placid existence, apart from the strife and turmoil of our civilization; an interesting corollary is found in the United States, where the negro yearly enters more and more into the competition of modern life, and yearly loses more of his freedom from *tabes*; indeed, a case of *tabes* in a negro there, once noteworthy, is now commonplace. The occurrence of *tabes* mainly among males and mainly among the intellectual classes also suggests that the mental stress of a modern environment or of an intellectual occupation may predispose the syphilitic to *tabes*.

We have already mentioned (p. 119) the presence of cell groups in the central nervous system, which are called centers of vegetative function. These centers correlate vegetative processes in response not merely to peripheral but also to psychic demands. For every psychic process there is a concomitant physicochemical equivalent. These equivalents are unconsciously evoked; thus, with shame occurs blushing, dilatation of the blood vessels of the face and neck; with fear, pallor—vasoconstriction—accelerated heart action, goose flesh and

relaxation of sphincters; and with intellectual effort a rise of blood pressure.

The rise in blood pressure during mental effort has been shown by Cannon and de la Paz to be accompanied by increased secretion in the adrenal glands. Cannon also has shown that in states of fear there is an augmentation in the amount of adrenalin in the blood.

The more frequent the rise of blood pressure due to psychic effort, the longer the pressure takes to resume the normal average in the resting intervals; the more does this resting average tend to rise; and the more do excessive rises result from mental effort. In other words, the more frequent or the more prolonged the mental effort, the more irritable is the sympathetic, the more is its normal balance with the vagus disturbed.

In syphilitics exposed to mental strain, slow and irregular heart action; nausea, hypersecretion and abnormal gastric movements; spastic constipation; and other symptoms characteristic of unbalanced vagal irritability occur. To test the vagal tone, clinicians inject 0.01gm., of pilocarpin hydrochlorid. An abnormal degree of sweating and salivation shows the presence of vagal irritability. Not only may vagotonia be thus revealed in syphilis, but a gastric crisis may sometimes be evoked, disclosing the presence of unsuspected incipient tabes (Hess and Eppinger).

Intellectual strain disorganizes the normal balance of tone between the antagonistic vagal and sympathetic components of the vegetative system; impairs in consequence that regulation by the vegetative nervous system of the chemical confederacy which ensures the defense of the body against spirochete invasion; and, therefore, renders the defense imperfect. In consequence the spiro-

chete persists to cause tabes. A virulent infection may bring tabes to the normal individual, but an infection of average virulence will bring it to the nervous. The nervous syphilitic is a potential tabetic, a potential general paretic. The liability of the tabetic to pneumonia, tuberculosis and other intercurrent infections, may be due in part to his disease; but it is due in part, also, to the imperfect defense to which the occurrence of tabes is attributable.

The imperfection of the tabetic's defense against the spirochete may be inherited; nervous lesions have been reported in members of the same family who were infected with syphilis from separate sources: it may be acquired owing to environmental or occupational conditions causing mental stress which disturbs the normal balance of the vegetative system; it may be due to the mildness or absence of the pretabetic lesions causing an inadequate production of defense substances, an inadequacy in the chemical stimulation of the vegetative nervous system; it may be due to the invincible virulence of the attacking spirochete. This imperfect defense enables the spirochete to persist active. The attacking force of the spirochetes, their number and their virulence, depends not merely upon qualities inherent to the spirochete, but also upon the efficiency of the defenses outside of the central nervous system. Cases with lesions of the vegetative, nervous system, which controls this defense, are, *ipso facto*, exposed to a greater attacking force than cases in which this controlling mechanism escapes.

In optic tabes the vegetative nervous system is seldom widely implicated; the central nervous system may thus confront only a few persistent spirochetes: whereas in spinal tabes not only may the vagal component be found

extensively degenerated, but the sympathetic fibers in the posterior roots are invariably attacked. A more or less continuous stream of spirochetes may be liberated from a quiescent resistant stage by the gradual deterioration which the general defense undergoes, as the control of the vegetative nervous system fails. The insignificance of the vegetative lesions and in optic tabes may be a factor in making that form the abortive form of tabes, the "*forme fruste par excellence*" (Fournier).

As tabes beginning in the posterior roots affects mainly posterior roots; as tabes beginning in one optic nerve next affects the other; and as the resulting optic degeneration is greater when tabes begins in the optic nerve than when optic atrophy follows spinal atrophy, the spirochete seems to grow best in sites which are chemically similar to that where it first grows in the central nervous system. The usual site first attacked in the central nervous system is, however, neither the posterior roots nor the optic nerve, but that part of the vegetative nervous system which supplies the pupil. Sometimes the disease spreads mainly in the vegetative nervous system: many cases of tabes complain first of bladder symptoms; some tabetics are troubled almost exclusively with gastric crises. But, usually, after affecting the vegetative nervous system of the pupil, posterior root lesions occur. The vegetative nervous system responds to chemical stimuli. Different chemical stimuli affect different parts of the system. There is, therefore, probably a lack of uniformity in the chemical composition of the several parts of the vegetative nervous system, a lack which may explain why the vegetative nervous system, although the first site, is not, as a rule, the main site of the spirochete's invasion.

CHAPTER VIII

DIAGNOSIS AND COURSE OF TABES

Symptoms and signs of tabes. Their origin. Diagnosis of syphilis. First sign of invasion of nervous system. Course of invasion beginning in posterior root; beginning in optic nerve. Tabes diagnosed by evolution. Tabes a disease in which dominant symptoms arise from primary syphilitic invasion of posterior nerve roots. Pretabetic period, duration, and significance. Effect of age on pretabetic period. Rate of syphilitic invasion. Rapid, slow and abortive invasions. Remissions. Preataxic stage. Ataxic stage. "Paralytic" or surrender stage. These stages psychological, not anatomical. Types of tabes. Age of onset of tabes. Termination of tabes.

THE observers named in the following table record the frequency of the signs and symptoms (Abadie signs only) which they have observed in tabes, as follows:

	Leimbach 400 cases	Abadie	Sarbo	Schaffer 200 cases	
				Incipient	Advanced
Absent knee-jerks	96.52%	70%	89.40%		
Absent Achilles jerks		82	91.00	14%	71.6%
Romberg's sign	88.75	..	93.00	25	73.3
Lightning pains	88.25	..	93.00	53	90.
Bladder trouble		65	79.00	14	63.3
Pupillary disturbances.		65	88.80	89	98.3
Peroneal anesthesia		80	88.50	..	
Ulnar anesthesia		..	66.00	..	

Of these, we have shown the pupillary, the bladder, and, in part, the lightning pains to be due to implication of the vegetative nervous system; the loss of tendon reflexes and the common sensory loss to be due to implication of sensory fibers in the posterior nerve roots;

and Romberg's sign to be due partly to sensory loss and partly to mental causes.

The vegetative, non-vegetative, sentient, and non-sentient fibers of the afferent nervous mechanism are thus implicated in the syphilitic invasion of the nervous system styled tabes. Its diagnosis rests upon evidence of syphilis and evidence of implication of the afferent nervous mechanism.

In all reactions of the tissues to organisms, in all inflammations, organismal toxins produce by chemotaxis a serous and a cellular exudate at the site of invasion. The tissue reaction to the spirochete in the central nervous system increases the bulk and the content of the cerebrospinal fluid. The increase of the bulk of the cerebrospinal fluid causes a raising of its pressure, which is evident in the greater force of its flow when the fluid is tapped by lumbar puncture. The increase in the contents consists mainly of globulin and of small lymphocytes. The amount of globulin varies. Apelt and Nonne, by careful investigations, have determined the amounts of globulin which commonly occur in tabes and have stated that the probability of the presence of tabes may be gauged from these amounts. The occasional cell seen normally in a microscope field of the centrifugalized cerebrospinal fluid is multiplied twenty to sixty fold in tabes. But more important than these inflammatory evidences is the presence in the cerebrospinal fluid of the substance which is practically pathognomonic of syphilis, the Wassermann substance. From the globulin content, the cell count, and the Wassermann reaction in the cerebrospinal fluid, the presence or absence of the spirochete in the central nervous system is presumed (*see Warthin's researches, p. 170*).

Mental stress is often a causal factor in bringing tabes to the syphilitic. The earliest symptom of syphilitic invasion of the nervous system may be neurasthenia. The future course of the syphilitic neurasthenic cannot be foretold. He may develop no further symptoms or he may become a paretic or a tabetic. In testing with atropin the vagal irritability of a syphilitic neurasthenic, Hess and Eppinger elicited a gastric crisis and discovered the presence of unsuspected tabes.

The earliest sign of syphilitic invasion of the central nervous system is the Argyll-Robertson pupil or its precursors. But variation in the shape or the size of the pupil or diminution of its contractility to light, is by none alleged to suffice for a diagnosis of tabes. An Argyll-Robertson pupil may be followed by tabes, by paresis, by optic atrophy, by amyotrophic lateral sclerosis, by any of the groups of symptoms which may follow syphilitic invasion of the central nervous system. It may also be the unique sign of an abortive invasion.

There is no agreement regarding the minimum that constitutes tabes. Lightning pains we have seen are due to irritation of the vegetative or of the common pain fibers usually in the posterior root. Root irritation may be caused by inflammatory processes, or by the pressure of a tumor or of an aneurysm. In a syphilitic, such pains occurring without evidence of root pressure usually arouse the first suspicion of tabes. Less than pupillary changes and lightning pains seldom suggest tabes. The first suspicion of tabes arises, therefore, from a syphilitic implication of the vegetative and the sensory systems. The single site at which such implication commonly occurs is the posterior root. Hence the possibility of tabes suggested by pupillary changes and

lightning pains becomes a probability when there occurs also a loss of tendon reflexes due neither to alcohol, nor to diabetes, and not associated with pain on pressure such as is seen in neuritis. If to this loss be added a loss of sensibility to pain greater in area than that of the loss to touch, the existence of a posterior root lesion becomes certain.

We have seen that with the exception of the vegetative system, the site first attacked is usually the chief site of the invasion, that syphilitic invasion beginning in the optic nerve affects it mainly; that when the invasion begins in the optic nerve, the destruction there is usually more severe than when optic follows posterior root invasion; that invasion beginning in the optic nerve may be confined there, whereas invasion beginning in the posterior root, although affecting mainly posterior roots, may later implicate any cranial nerve and may become almost coextensive with the central nervous system. A primary optic lesion is thus associated with a more or less characteristic invasion; so also is the primary posterior root lesion.

We have seen that the vegetative nervous system controls the chemical confederacy to which defense against organismal invasion is entrusted. Lesions of that system may permit weakly spirochetes to persist, may allow inactive spirochetes to become aggressive, may entail successive streams of organisms sensitizing and later invading the nervous system. Posterior root lesions imply vegetative lesions. Optic nerve lesions do not. Hence the initial site of the invasion somewhat presages the course.

The syphilitic posterior root lesion does not make the presence of tabes certain. Paresis remains paresis in

spite of root lesions. Syphilitic root lesions in aortic disease are not considered as tabetic. Root lesions may occur in cerebral gummata. It is recognition that the source of the dominant symptoms is primary syphilitic invasion of the posterior roots which leads to a diagnosis of tabes. When symptoms are few, we wait till the certainty of a posterior root lesion can be established. When symptoms are many, we ignore the posterior root lesion if, as in paresis, it does not dominate the disease picture. It is the course, not the site, of the invasion which we strive consciously or unconsciously to determine. We diagnose syphilitic invasions by evolution.

Regarding the diagnosis of "l'ataxie locomotrice progressive," Duchenne states:—

"What really makes the ataxia a distinct, morbid entity, is the regularity in general of its mode of appearance and of the development of the symptoms; it is their sequence, in other words, their order. Ataxia can be divided into three distinct periods; the first characterised by the paralysis of one or several motor nerves of the eye complicated by the paralysis of the optic nerve and by pains, shooting, erratic; the second, by the appearance of troubles of coördination usually in the lower limbs but sometimes in the upper; the third, by the generalization of the malady."

There is no pathognomonic sign of tabes. We must still, like Duchenne, base the diagnosis upon evolution. But the discovery by Argyll-Robertson of Edinburgh (1869) of the loss of light reaction in the pupil of syphilis; by Westphal (1875) of the loss of the knee-jerks; by Erb of the swaying when the tabetic stands with closed eyes (Romberg's sign); by Wassermann of the serological evidence of syphilis; and by Head and Sherren of the nature of the sensory loss in root lesions,

enables us to recognize tabes without waiting so long as Duchenne.

The period between the causal syphilitic infection and the assured advent of tabes constitutes the pretabetic period. At least twenty per cent of tabetics do not know they have syphilis. Others have such mild pretabetic lesions that they are ignorant of the date of their infection. Grossman¹ found among 240 cases only 103 with a history of a primary sore. The length of this pretabetic interval, erroneously styled the incubation period, is therefore usually impossible to determine with accuracy. The duration of the pretabetic period depends on the attacking force of the spirochete and on the weakness of the resistance of the nervous system. The greater the attacking force of the spirochete and the weaker the defense, the shorter is the pretabetic interval. The duration of the pretabetic interval may vary from a few months to fifty years. Pauly treated a person for a chancre on Sept. 25, 1891, in a public hospital in Lyons (*Lyon médicale*, June 12, 1892). On Dec. 21 the patient showed mucous patches and double iritis. On Jan. 6, 1892, laryngeal crises occurred. On Jan. 25 difficulty in standing was evident and on Feb. 8, about five months after infection, ataxia of the legs appeared. Fournier records a case of syphilitic infection in Jan., 1881, followed in March of the same year by Romberg's sign, lightning pains and diminution of vision.

Besides these fulminating cases, tabes beginning

¹ The 240 cases of tabes analyzed by my assistant, Dr. M. Grossman, comprise material from the Mt. Sinai Hospital kindly placed at his disposal by Dr. B. Sachs; material from the Montefiore Home, available through the courtesy of Dr. Wachsmann; and material from my service in the Central and Neurological Hospital, New York City.

twenty-five to thirty years after the incriminated infection occurs. Dieulafoy (*Gazette hebdom.* Aug. 31, 1877) reports a case of syphilis in 1830, followed by lightning pains in 1865—in the patient's sixty-eighth year—and by diplopia and ataxia in 1870. Inglerans (*loc. cit.*) saw a tabetic who, in 1861, at the age of twenty-six, acquired syphilis and who had lightning pains first in 1895. The pretabetic period in the last two cases was thirty-five and thirty-four years respectively. Among Grossman's 240 cases, the pretabetic period ranged between 25 and 32 years in thirteen cases.

Lowinsky's 70 cases had an average pretabetic period of 13 years. In 43 private cases in which the infection could be accurately dated, I found the average pretabetic period was 11.3 years. Grossman's 240 cases comprised 136 who were ignorant of, or uncertain about, their infection; the 103, who provided a precise history of chancre, had an average pretabetic interval of 14 years. As there is no agreement upon what constitutes the minimum essential for tabes, and as tabes may long be latent before the patient seeks the physician, even the lowest of these figures, 11.3 years, is probably too high.

It is affirmed that the shorter the pretabetic period the more unfavorable is the prognosis. Von Malaisé asserts that a pretabetic period of less than six years is ominous. Grossman's series comprised 13 cases showing pretabetic periods of six years and less. The average pretabetic period of these 13 was 3.9 years; none of these 13 has died; their average age is 44 years; the disease has endured among them on an average 15.3 years. A short pretabetic period may be, but is not necessarily, an unfavorable sign.

Erb states that the younger a person acquires syphilis,

the quicker tabes follows. He reports a case of infection at 19½ years followed by tabes at 22. In support of this statement, Mingazzini and Salvadori record an infection at 18 years followed by shooting pains at 19½ years; and Schaffer publishes a case of infection at 21, leading to tabes at 23. The shortest pretabetic period in Grossman's series was 11½ years and occurred in a man aged 40½ years. Grossman reports 25 cases of tabes which referred their infection to chancres received between the ages of 13 and 20. The average age of infection of these 25 was 18.44 years; among them the pretabetic period varied from 2 to 31½ years; and averaged 16.6 years. Statements regarding the influence of age on the pretabetic period should be accepted with reserve until tables showing the incidence of syphilis and tabes at all ages are available.

The rate at which the syphilitic invasion called tabes spreads varies profoundly. Fournier's and Pauly's cases of rapid generalization have just been mentioned. Vulpian records a case in which generalization occurred in four months; Rosenthal, in five months; Trousseau, in six months; and Buzzard, in fifteen months. I had under my care at the Central and Neurological Hospital a case who first sought advice for laryngeal crises; he had then no knee-jerks, and slight sensory loss in the lower limbs; next his vision failed completely in one eye, partially in the other; then he developed general paresis. This course occupied eleven weeks.

In contrast with these rapid cases is a tabetic (reported by Raymond) who suffered from lightning pains, dysuria and diplopia at the age of forty-one and who, with no knee-jerks and fixed pupils, still pursued his non-ataxic way at the age of eighty-six. Literature is crowded with

reports of cases in which the invasion never spread. Thus, Déjerine reports three undoubted tabetics who suffered for thirty, thirty, and thirty-six years respectively, only from pain: Fournier, one who for twenty-three years, Heldenbergh, one who for eighteen years, and Vulpian, one who for ten years, suffered only from crises. Raymond reports a tabetic who suffered only from blindness for forty-three years; Förster, one merely blind for thirty-three years. Schuster found seventy-eight abortive cases among five hundred tabetics. Our ideas of the spread of tabes have all been clouded by confounding the progress of the structural changes with the mental deterioration which results from them.

Besides more or less permanent arrest of the invasion, prolonged periods of quiescence may occur. During these periods of quiescence, the mental element in symptoms may spontaneously lessen. These periods are hence termed remissions.

The interval between the onset of tabes and the onset of ataxia is called the preataxic period. We have seen that the onset of tabes cannot be absolutely defined and that the onset of ataxia, as it usually is insidious, is also difficult to date. The duration of the preataxic period is extremely variable. Fournier reported 65 cases among which the preataxic period lasted two years or less in 19; more than two years but less than five in 23; and from five to fourteen years in 23. In fifty-four per cent of Fournier's cases the preataxic period was three years or less.

In 59 per cent of 236 cases of Grossman's series the preataxic period was three years or less.

Grossman's figures are as follows:—

No. of cases	Interval between the ascertainable onset of tabes and the appearance of ataxia.
60	Less than 6 months
17	6 months to 1 year
24	1 to 2 years
27	2 to 3 “
22	3 to 4 “
20	4 to 5 “
19	5 to 6 “
10	6 to 7 “
12	7 to 8 “
5	8 to 9 “
3	9 to 10 “
4	10 to 11 “
2	11 to 12 “
1	13 “
3	14 to 15 “
7	15 to 27 “

The preataxic period is alleged to be shorter in the young and in the old than in the middle-aged. If we consider as young those in whom tabes begins at or before the age of 30; and as old, those in whom tabes begins at or after the age of 50, Grossman's series shows 35 young of whom 11 had a preataxic period of 1 year or less; 33 old of whom 16 were preataxic for 1 year or less, and 170 between the ages of 30 and 50, of whom 57 were preataxic for 1 year or less. The preataxic period was 1 year or less in 31 per cent of the young; in 48 per cent of the old, and in 31 per cent of the remainder.

The preataxic period is prolonged in the blind. Cases, at my disposal, of ataxia following blindness are too few to permit any estimation of the duration of the preataxic period in the blind. Statistics of the duration of the

preataxic period have little absolute value. Fournier has published a further list of 86 cases in which ataxia had not yet appeared. The duration of these cases ranged from 1 to 30 years; the average duration was 5.7 years. Cases of forty and of fifty-five years' duration without ataxia have been recorded.

The ataxic stage dates from the onset of ataxia to the onset of helplessness. It is said to last from ten to twenty years. Most tabetics perpetuate the ataxic stage and never become bedridden.

Grossman's series comprises 36 so-called "paralytic" cases—chair- or bedridden tabetics.

Six cases had been immobile for one year or less; six for more than one but less than three years; twelve for three to four years; four for more than four but less than eight years; four for eight to twelve years. Twenty of these cases had been immobile from three to twelve years. The duration of the immobile period is determined mainly by the nurse.

The remaining four of these immobilized patients died; one at the age of 30, after four years of bed existence, from pylonephrosis and pneumonia; another aged 39, after three months in bed, from pylonephrosis; a third, 50 years old, bedridden for 12 years, from arteriosclerosis; a fourth, aged 49, after seven months immobility, from an unascertained cause.

Among these immobile tabetics there are three main groups; one, containing 16 cases immobilized by ataxia complicated by other factors, such as joint trouble and fractures; another, containing five non-ataxic cases immobilized by blindness or injuries, and a third containing 15 cases immobilized by ataxia alone.

Among the 16 ataxics forming the first group, immo-

bilization followed in 5 cases the concurrence of blindness and ataxia, and in 5 others, the appearance of Charcot joints; 2 cases were attributed to fracture and Charcot joints; 1 to fracture without joint trouble; one to intraspinal medication; one to perforating ulcer in the foot, and one to muscular atrophy, complicating ataxia. The tabetic who blamed intraspinal medication for his bedridden state was aged 72. After 5 years contending with gastric crises, he became ataxic. He persisted active for 7 years more. Intraspinal medication was his last hope and when it failed he gave up the struggle. Only in a single case, which was bedridden because of muscular atrophy, could a real paralytic stage be distinguished. Of 5 in the second group none have had ataxia: three took to bed and remained there after injury to their lower limbs; 2 after the onset of blindness. Of the 15 immobilized because of ataxia alone, 2 retired to bed immediately ataxia appeared. Among the 6 who became bedridden within 4 years—six, six, twelve, eighteen, twenty-four and forty-two months after the appearance of uncomplicated ataxia—the pre-ataxic period was 2 years or less in 5.

The insatiable instinct "to distinguish" which the physician shares with—perhaps, inherits from—the scholastic, has led to the differentiation of myriads of types of tabes. We distinguish pain types, crises types, bladder types, ataxic types, blind types and deaf types, sensory types and motor types, tardy types and fulminating types, juvenile tabes, adolescent tabes, senile tabes, spinal, sacral, lumbar and dorsal tabes, cervical, bulbar and optic tabes; and so forth endlessly.

From the unreliable point of the appearance of the first symptom, the age at the onset of tabes is calculated.

It has no limits; tabes may appear in childhood and in adolescence as well as in middle age. I have seen two in whom tabes first appeared after their fifty-fifth year. Several over 70 years old have been recorded.

Moebius, Karger, Berger and Friedrichsen record a total of seventy-four tabetic women among whom tabes occurred in sixty-six (89 per cent) before the age of fifty; and in all before sixty. Under twenty-five years of age, seven cases occurred; after fifty, eight; and between twenty-five and fifty, fifty-nine. Of one hundred and eighty-five cases recorded by Erb and Karger, two per cent occurred before twenty years of age; ten per cent before thirty; sixty per cent before forty; and ninety per cent before fifty. Tabes, therefore, occurs usually between the ages of thirty and forty-five; and is rare after fifty and before twenty-five. The age incidence of tabes in Grossman's series has already been mentioned.

To estimate the average age of onset of tabes we must ignore the rare cases, the cases after fifty and before twenty-five. Of the Moebius series the average¹ age at onset was thirty-five and a half years; of Erb and Karger's series thirty-eight years; of seventy-eight cases which I saw within seven years of the onset of the disease, 36.9 years; of 239 cases collected by Grossman 39.1 years.

The average age at death in 33 out of the 34 cases reported by Burr was, 53.46 years; in 19 cases, 57 per cent, death occurred at 53 years or later. The average age at death of the 28 cases in Grossman's series, was also 53 years: in 15 cases, 53 per cent, death occurred at 53 years or later. In Grossman's series there were 77

¹ All averages are arithmetical means, except those of my cases which are *probable means* (*Wahrscheinliche Mittel*).

tabetics aged 53 years or more. The death rate among tabetics of 53 years or more was therefore 195 per thousand.

The causes of death in tabes are two—cardiovascular disease, and adventitious infections, such as cystitis, pyonephrosis, pneumonia and tuberculosis. The spirochete attacking the central nervous system produces simultaneously less obvious but sometimes more grave lesions in the cardiovascular system; and such lesions may be aggravated by the massive doses of the metallic poisons, mercury and arsenic, which are used to combat the spirochete. The concurrence of syphilis and tubercle is always menacing; there exists no proof that tuberculosis is more lethal to the tabetic than to any other syphilitic. The liability of the tabetic to tuberculosis has not been proved to be greater than that of the non-tabetic syphilitic. But an imperfect resistance which renders the central nervous system liable to attack may make tabetics prone to intercurrent affections and the resistance may be still further reduced by the vegetative lesions of tabes.

None ever dies of tabes, just as none ever dies of blindness, or of deafness, or of other sensory defect.

The lethality of tabes is that of syphilis. Raymond's tabetic, hale and hearty at the age of eighty-six; and Trousseau's octogenarian tabetic, possessed of a "*vig-ueur insolite a son âge*" show how benign tabes may be.

CHAPTER IX

ANTISYPHILITIC TREATMENT IN TABES

Essential tabetic lesion irreparable. Tabes incurable. Effect of antisyphilitic treatment on the duration of the pretabetic period. Absence of proof that such treatment can prevent tabes. Nature of the evidence required to establish such preventive action. Absence of proof that antisyphilitic substances arrest tabes. Nature of the evidence required to establish the power of a drug to arrest tabes. Drugs commonly used in tabes. Mercury: modes of administration. Arsenic, enesol, salvarsan. Motives for intraspinal administration unfounded. The search for new preparations of antisyphilitic substances and for new methods of administration: indications of the inadequacy of these substances. Necessity for caution in the use of mercury and arsenic in tabes. Nucleinates and nucleinic acid.

THE fundamental lesion of tabes is a degeneration of the nerve processes. Whether this degeneration occurs in the spinal roots, or in the optic nerve, or elsewhere, it is not secondary to meningeal inflammation but is a result of invasion of the nerve processes themselves. Any meningeal inflammation which occurs is a concomitant, a more or less accidental complication of the invasion of the nerve processes (p. 20).

Tabetic degeneration occurs essentially in nerve processes which lack the protection of the neurilemmal sheath, the sheath of Schwann. It may be laid down as an axiom that nerves in which Schwann's sheath is absent, nerves such as constitute the white matter of the central nervous system, when once degenerated, do not spontaneously regenerate. Any medication which cannot be demonstrated to have the power to regenerate nerves,

cannot cure a disease in which degeneration of nerves occurs. A considerable remedial influence can be exerted upon the inflammatory exudates which accompany syphilitic invasion of nerve processes and upon the proliferation in the meninges—when such exists—by means of iodids, mercury, arsenic and other antisymphilitic remedies. As objective evidence of this influence, we find a change in the nature of the Wassermann reaction, and a decrease of the cellular elements in the cerebrospinal fluid; exceptionally, a less sluggish tendon reflex, a less fixed pupil: as subjective, a diminution in some symptoms, particularly pain. Apart from the removal by medicinal means of exudates and of the syphilitic tissue overgrowth, which in the meninges may impair function, or even cause destruction of the reflex paths, it is certain that we can by no measures now at our disposal repair the affected structures in tabes. As we know no substance which will cure the nerve degeneration which is the essential lesion of tabes, we know no cure for tabes.

Inquiry elicits that some tabetics receive no antisymphilitic treatment for their pretabetic lesions; that others receive merely cursory treatment; and that others again receive what even the captious must consider adequate. No proof was ever offered that the non-tabetic receive more antisymphilitic treatment than the tabetic. Many pretabetics receive cursory treatment because of the trivial nature of their syphilids. These trivial syphilids are evidence of a trivial reaction to the spirochete and of a trivial production of defense substances. This trivial reaction may be adequate to kill the spirochete: some syphilitics have but mild syphilids and have no subsequent symptoms. But others owe to the mildness of the pretabetic lesions the inadequacy of the defensive

substances produced by the outpost tissues, which leads to the persistence of the spirochete and later to the development of tabes.

Noehte states that pretabetic antisymphilitic treatment has no influence upon the later appearance and course of tabes. Lowinsky reports that the average interval between the primary syphilitic infection and the first symptom of tabes in a series of tabetics which he investigated was as follows:—

A. In untreated cases (14 men, 1 woman), fourteen years.

B. In cases inadequately (*unzulänglich*) treated (39 men, 10 women), thirteen years.

C. In cases fully (*ausreichlich*) treated (5 men, 1 woman), eleven years.

Lowinsky unexpectedly makes these statistics a basis for a plea, not against, but on behalf of, energetic antisymphilitic treatment in the pretabetic. Lowinsky's figures must not be unduly emphasized. I have found no difference in the age of onset of tabes among those who were cognizant and those who were ignorant of their syphilitic infection; among those, in other words, who presumably were treated and those who were not.

As tabes does not necessarily follow untreated or cursorily treated syphilis; as tabes may occur in spite of intensive antisymphilitic treatment; and as antisymphilitic treatment may not lengthen, and may even be associated with a shortening of, the pretabetic period, the value of antisymphilitic treatment in preventing tabes needs proof.

To ascertain if we can prevent tabes by treatment, we must know how often tabes occurs in untreated and in treated syphilis, respectively. The civilized are subject to and the primitive are free from antisymphilitic treat-

ment and tabes. Too many other differences distinguish civilized from primitive races to permit any conclusions regarding the causal relation of antisyphilitic treatment to tabes to be drawn from this fact. In the same race the ratio of tabes to syphilis varies according to sex, environmental, occupational and constitutional conditions. To ascertain the effect of treatment in preventing tabes we must eliminate as many as possible of these apparent sources of variation. If investigation be narrowed to males of the same race, age, social condition and occupation, the larger the number considered, the more will any other sources of error, such as differences in individual resistance and in the virulence of the infecting spirochete, be discounted. The ideal field for such an investigation is the services. There, competent observers have care of large bodies of men who are approximately of the same age, civilization and social standing; who are similarly employed; who, if syphilitic, are skillfully, equally and assiduously treated; and who can be observed for years. The frequency of syphilis among these men should be ascertained by blood examination. Vedder has already made a notable beginning by examining the United States recruits. If from the services we can only ascertain the frequency with which syphilitic disease of the nervous system occurs in treated syphilis, we shall be able to compare with that the frequency in analogous groups of less treated civilians. Even from this evidently inaccurate comparison we shall gain some knowledge of the power or impotence of accepted antisyphilitic remedies to prevent invasion of the nervous system. At present we have no knowledge: only unverified tradition and belief.

To prove that a drug can arrest tabes, we must know

the rate at which tabes advances. Few things are, however, so variable as this rate. Tabes may advance with fulminating rapidity; may progress insidiously and this progress may be punctuated by sudden exacerbations and remissions; or may commence, advance slightly, and then spontaneously stop. Schuster found seventy-eight abortive cases among five hundred tabetics.

Therefore, in a given case, we cannot foretell the rate of progress in tabes, nor may we affirm the causal relation between the administration of a drug and the arrest of the tabetizing process. If we could observe a sufficiently large series of cases of tabes for a period wherein definite advance in the lesions of tabes might be expected, and if of this series half of the cases were reserved as a standard, and were given no medication, while the other half were treated with the alleged remedy, we could reach conclusions which, although not permitting unrestricted generalization, yet would be of notable value. In the absence of such data, a physician who states that a drug has arrested tabes relies on a prognostic acumen which is denied to his fellows: his statement exposes him not only to credit for supposed success but also to blame for failure.

A substance averred to arrest tabes must be able to stop the advance of tabes. It is upon the failures to stop advance, not upon alleged arrests, that judgment of remedies in a disease of such uncertain course as tabes should be based. Every neurologist observes the failure of antisyphilitic remedies to prevent the advance of tabes with a frequency which is greater the larger his practice in tabes. Déjerine could detect no influence of mercury, and I no influence of salvarsan, on tabetic optic atrophy.

Professor Warthin by a long and careful study was able conclusively to demonstrate post mortem in a series of cases of tabes and other forms of old syphilis the presence of the spirochete in the tissues, not merely of those who had received intensive antisyphilitic treatment, but even in those who because of that treatment had attained before death a negative Wassermann reaction.

We know no drug which can cure tabes, and we are uncertain of the power of antisyphilitic remedies to prevent or to arrest tabes. Our reaction to this ignorance is reflected in the drug treatment of tabes. The more unsatisfactory the medicinal treatment of a disease proves, the more numerous are the drugs lauded as efficacious. When we possess a specific for a disease, as quinine for malaria, it reigns alone. There are few drugs in the pharmacopoeia which have not been invoked to aid the tabetic. Those which have been most used are silver, mercurial and arsenical preparations, and the alkaline iodids. Silver salts were fashionable until the frequency of argyria made them lose favor. Iodids were employed to aid the absorption of the inflammatory products of syphilis, but, as they were not dramatic in action and as they habitually were given in doses so small that little reached the cerebrospinal system, and all was speedily excreted, they fell into unmerited disuse.

With the growing belief that syphilis caused tabes, all except antisyphilitic substances ceased to be employed. Mercury was long the chosen drug. It formerly was given by the mouth. Inaccuracy of dosing and other disadvantages led to oral administration being replaced by inunction. Inunction combined quick absorption of

the mercury not only with the useful measures of massage and bathing, but also with the psychic influence of the rubber. These rubbers, especially at Aix-la-Chapelle, where they constitute a class with almost hereditary privileges, were skilled to exert a beneficent mental effect upon the tabetic, the most suggestible of all patients. Added to the mercury, bathing, massage, and rubber, was the psychotherapeutic influence of the spa. The distracted tabetic removed from his usual environment, was disciplined; his régime was regulated; the spa physician was a health-giver whose dictum was law; and the tabetic, in salutary competition for health, strove with the similarly sick. No device was missing to reinforce the psychic effect of mercurial inunction: so inunctions conferred, and still confer, benefit.

Inhalation was offered unsuccessfully as a rival to inunction, which, however, was later somewhat displaced by injection. As injections proved too irritating when subcutaneous, they were made intramuscular. Then, to avoid soaking all tissues in mercury, in order to induce with the surplus an influence upon the central nervous system, intraspinal injection was introduced. At first careless experimenters, forgetting the irritation that followed even subcutaneous injection, used salts, such as the perchlorid, which precipitate the albumen of any tissue they encounter and act as destructive agents at the site of the injection. Success did not follow, and the next step, direct administration to the diseased part, was made by Horsley. I occasionally assisted him when he trephined for gummata and laved the meningeal surface with perchloride of mercury.

During the greatest vogue of mercury, arsenic in one or other of its pharmacopoeial preparations was also used,

sometimes alone, sometimes in combination with iodids, or with mercury. Then proprietary compositions, such as enesol, were in turn devised, lauded and forgotten. Such was our consciousness of the failure of these substances that Ehrlich's announcement of salvarsan was welcomed almost universally. The enthusiasm of the protagonists of salvarsan inadvertently created a cult which was ludicrously like that created by the two thieves in Grimm's fairy tale who sold silk which remained invisible to all who were unworthy to exercise the function of the position which fate had bestowed upon them. Salvarsan began its clinical career as an injection, usually intravenous. Its lethality to the spirochete was said to be so much greater than its toxicity to human cells that by its aid the destruction of every spirochete in every nook of the body was, at first, considered feasible. The extravagance of these initial hopes soon remained only with the laity. Powerful and speedy as was the action of salvarsan upon pretabetic lesions, it proved perhaps even less permanent than the action of mercury. McDonagh suggested that the spirochete has an inactive, resistant, spore-forming stage in which it is impervious to salvarsan.

The failure of salvarsan in syphilitic nervous lesions was admitted by Ehrlich when he stated that salvarsan was an adjuvant to the human defensive substances, and that defensive substances were present in the blood but absent from the cerebrospinal fluid.

Meanwhile, Willcox claimed that after intravenous administration of salvarsan, arsenic occurred in no considerable quantity in the brain, although plentifully present in other organs, such as the liver and kidneys; and Camp, that it was missing from the cerebrospinal fluid.

So therapeutists injected salvarsan intravenously; after forty-five minutes, withdrew blood from the injected person; and then introduced the salvarsanized serum intraspinally. Thus they ingeniously placed a minute dose—about 0.2 milligrams—of a benign form of salvarsan, plus the necessary defensive substances, in contact with the diseased tissues in the cord. Benedict now proves that the intravenous produces a greater concentration of salvarsan in the cerebrospinal fluid than does such intraspinal administration. He injected salvarsan intravenously and fifteen to forty-five minutes later, estimated its amount in the blood. After twenty-four hours he found salvarsan not only present in the cerebrospinal fluid but present still in one-sixth to one-tenth the concentration he first found in the blood.

Yet many never permit themselves to realize that these drugs frankly fail in tabes; only the method of using them may be acknowledged faulty; hence, new methods of administration are invented and the life of the false specific is artificially prolonged by fresh groups of the credulous. Thus, there is now appearing in lay and in medical journals an attempt to replace needless intraspinal, by intracerebral, or by intraventricular injection of salvarsan and of mercury. Those who calmly advise such treatment ignore the mortality inseparable from trephining; ignore the prodigious shock of the operation to those who survive; ignore the fact that direct application of the drug to the spinal cord failed to arrest the syphilitic lesions there; and in their reports sometimes show ignorance even of the principles which should govern their investigation, if their dangerous procedure be meant to have any experimental value.

The lack of success of antisyphilitic substances is ac-

knowledge in the incessant changes which are made in the form and in the method of their administration. Yet all are agreed that their failure is not absolute. We sometimes achieve slight, if temporary, success in the relief of tabetic symptoms by these substances. The aid is not wholly due to the drug: the physical effects of massage and baths in mercurial inunction and of lumbar puncture in intraspinal injections; and the mental influence inseparable from all medicinal treatment, must be credited with some share in any improvement which results. The rest of their aid is mainly due to the so-called alterative effect, the effect upon the vegetative nervous system, which arsenic, mercury and iodids produce; and to the solvent action of these drugs upon meningeal and interstitial exudates, the accidental and non-essential accompaniments of the syphilitic invasion of nerve processes.

In default of more certain remedies, we must still avail ourselves of any aid which these substances lend. But the limitations of such aid should be clearly recognized. We should realize that mercury and salvarsan, so valuable in removing banal syphilitic lesions, have not been proved to have demonstrable value in syphilitic lesions of the essential cells of usually sheltered tissues. Renal, hepatic, cardiac, vascular, and nervous parenchymatous lesions of syphilitic origin are all resistant to antisymphilitic remedies. Their obstinacy to treatment is due in part to the restricted capacity of the essential cells of these highly specialized tissues to repair and to regenerate. Mercury and arsenic are powerful protoplasmic poisons. They are generally considered to be contraindicated in non-syphilitic cardiac and renal diseases. Cardiac and renal diseases not infrequently bring

death to the tabetic. The nebulous aid of these substances in tabes should, therefore, be sought as cautiously as it is now sought recklessly.

Nucleinic acid and nucleinate of soda have been much lauded, especially in paresis. They produce a leucocytosis which is mainly mononuclear. After each injection, a transient febrile reaction occurs. Gradually an immunity to the drug develops, so that greater and greater doses are necessary to induce any reaction. The aim in treatment is to prolong the reactive period as much as possible; hence, small doses are used at first, and an increase is made only when the temperature and the blood count show that the dose last used is no longer capable of producing a characteristic effect. Strict aseptic precautions are necessary to prevent the occurrence of inflammation at the site of injection. I have used the nucleins both with and without the massive doses of salvarsan now advocated. In fourteen cases in which the nucleinate of soda was used, for several months, in which immunity to doses as high as two grams was achieved, I heard great reports from patients, but saw neither objective nor persistent subjective improvement in tabes. Indeed, one of my tabetics developed paresis after a three months' course of nucleinates, and quickly died.

CHAPTER X

THE MENTAL STATE OF TABES

Mental stress a cause of tabes. Tabes a source of mental stress. Tabes a cause of fear, of fatigue, and of depression. Psychoses in tabes. The psychosis typical of tabes an aggravation of the mental state induced by the tabetic symptoms. Resemblance between tabetic psychoses and prison psychoses. Measurement of the mental state of the tabetic. Weakening of attention in tabes: its sensory and motor effects. Variations in the mental state of tabes. Mental deterioration: its rate and its consequences. Influence of the tabetic symptoms upon the mental state. Influence of the mental state upon the tabetic symptoms. Estimation of the reciprocal relations of the tabetic symptoms and the mental state. Interdependence of tabetic symptoms. Foundations of mental treatment.

THE ratio of tabes to syphilis is higher in civilized than in primitive peoples, in men than in women, in the intellectual than in the working classes. A factor in determining this incidence is mental stress.

Life with tabes is exhausting. To endure pain, crises, and other manifestations of tabes taxes the strongest; to work in pain and fear and depression requires strenuous effort; to move with incoördinate limbs is a herculean task. The effort demanded by day soon exceeds the recuperation gained in the painful, sleepless nights. Slighter and slighter efforts, either mental or physical, fatigue the tabetic, quicken his pulse and respiration, cause palpitation and breathlessness.

The many perils to which the ataxic is exposed become increasingly harassing. His incapacity fills him with anxiety regarding his business and the sustenance of his family. To these worries, he adds, in many cases, fear

lest his wife or his child acquire the disease. He dreams of failure, of fires, of drowning, of starving on a raft in the midst of a limitless sea, of all kinds of catastrophes. Fear complexes, as they elaborate, more and more immediately succeed one another, until they produce an unrelieved state of perturbation. A veritable anxiety neurosis develops and extravagant manifestations of fear arise from the most trivial occurrences. Thus, the tabetic may show dread of the dark, of the open, of change of level, of change of surface, of crossings, of traffic, of strange places, of unaccustomed furniture, of company other than that of his family.

As the tabetic is attacked by one after the other of the unexpected, bewildering and mysterious manifestations of tabes, he loses heart. Unenlightened treatment fails to relieve him. The disease obviously and relentlessly advances. He feels increasingly his lack of competence to cope with his business, social and domestic affairs, and he becomes more and more depressed. This depression deepens till trivial impersonal occurrences, such as a gray day, produce altogether disproportionate results. Depression occasionally leads to drink, suicide, or an insane asylum.

The tabetic, fearful, irritable, depressed, weary, sleepless and emaciated, becomes less and less competent. He increasingly distrusts his sensations. He is, therefore, more and more shut off from his environment. Deafness and failing vision may add to his misery.

Everyone has a definite degree of ability to withstand mental strain. The degree varies; some are so constituted that they cannot long tolerate even the average stress of modern life. The limit of endurance may be passed suddenly as the result of a shock, or gradually,

in consequence of prolonged fatigue. Beyond the limit of endurance psychoses develop. The psychoses of tabes have been recorded by Meyer, Rydlewski, Henderson and others. They report cases of mental disease accidentally occurring in tabes, together with cases which are due to tabes.

Of fifty-four psychoses encountered in tabetics by Meyer, twenty-one were chronic hallucinatory paranoidias; fourteen, depressive psychoses; six, dementias; five, hallucinatory conditions; four, circular insanities; and the others were isolated examples of sundry psychoses. Rydlewski, in twenty-nine cases, saw paranoiac psychoses thirteen times; mania, four times; manic-depressive insanity thrice; hypochondria and melancholia twice; together with seven other forms of mental disturbances. Of Henderson's five tabetics, two suffered from hallucinatory conditions; and three from depression. Meyer states that there is nothing peculiar or characteristic in these psychoses. I think he errs. Psychoses may accidentally complicate tabes as they may complicate any disease. But the most frequent, the typical psychosis of tabes, is an aggravation of the mental state induced by the tabetic symptoms and habitually present in tabes; thus, the depression may be aggravated to a persistent hypochondriasis; fear may breed an anxiety neurosis; and living with deceptive sensory intelligence may lead to hallucinatory and to delusional insanity.

I have seen two tabetics under restraint showing no memory loss, nor other symptoms suggestive of general paresis, but delusions of hearing, extreme perturbation, fear of approach and of unseen influences; phases of restlessness alternating with periods of depression and with, in one case, catatonia. This psychosis strongly re-

semples that seen sometimes in the deaf and in prisoners undergoing solitary confinement.

We cannot measure the abnormality of the mental state for we have no standard of the patient's normal average. But from comparing his alleged former with his present business and social capacity, we judge the extent of his deterioration; and we can measure his present capacity for simple mental operations such as memorizing, counting, choosing, perceiving, associating, etc. A number of tests should be made so as to insure that

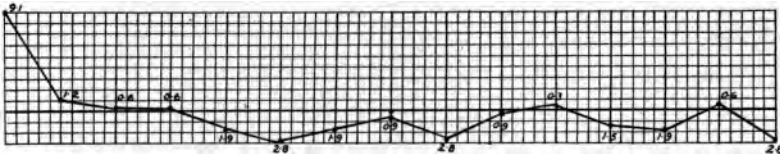


FIG. 67.—The reckoning test: Average curve of seven university graduates, showing from minute to minute the percentage variation from the average rate per minute on the third day of the test. The average total number of additions for the fifteen minutes was 951.

transient conditions of freshness, fatigue, interest, boredom, novice-ship and practice will not mislead us. The average of all phases of mental activity thus obtained affords a useful standard by which progress may be judged.

I have examined the capacity for slight mental work in thirty tabetics by means of the reckoning test.¹ The reckoning test I used is a modification of that devised by Oehren, and so brilliantly utilized by Kraepelin and his school. It consists of a book, resembling the familiar school copy book, every page of which contains digits, arranged in parallel vertical columns. The space between every two successive digits in a column is equal;

¹ Obtainable from the *Journal of Nervous and Mental Diseases*, 64 West 56th St., New York City.

between adjacent columns an uniform interval exists; and the margins are of adequate width. Every page has ten columns, each of twenty digits. The changes which I made in the arrangement of the text rendered the ease of writing as constant as possible throughout the page and enabled the number of pairs added to be readily ascertained. To economize space and gently to complicate the task, the digits are added in pairs *continuously*; i. e., after a pair of digits has yielded a sum, the

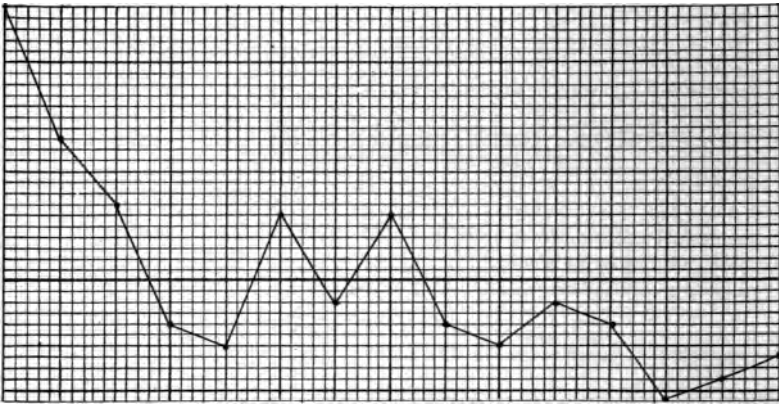


FIG. 68.—The reckoning test of A. R., tabetic. Curve showing percentage variation of work done from minute to minute on third day of test. Note the height of the initial spurt, the rapid falling off, the fluctuations from the fifth to the ninth minutes, and the subsequent fatigue. Marked fluctuations and early fatigue are due mainly to attention defects. Total output in fifteen minutes 521.

second digit of the pair is linked with the succeeding figure in the column, to form a new pair. Each digit (excepting the first in the first column) is thus used twice, for the last digit of one column is paired with the first of the next. Twenty sums are therefore obtained from every column of twenty digits.

The test is conducted as follows: The person to be tested is instructed in the method of *continuous* addition.

A few seconds' warning is given: then the signal to start. The person adds the digits in pairs as rapidly as he can. At the end of each minute the examining physician calls "Stroke"; the person at once makes a mark under the last sum he has written and proceeds immediately with the addition. By counting the sums between the marks, one can ascertain how many acts of addition, how many

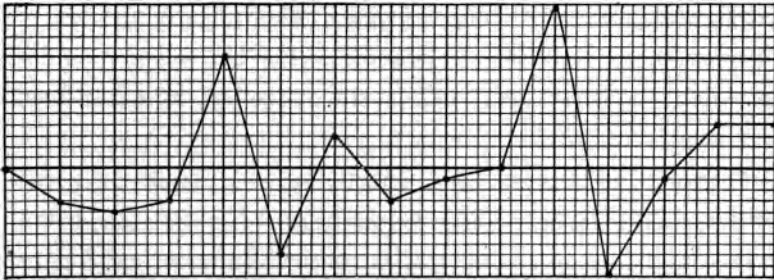


FIG. 69.—The reckoning test of J. H., tabetic. Curve showing percentage variation of work done from minute to minute on the third day of the test. Note the complete absence of the initial spurt, the sudden fluctuations, and the sustained increase in the last three minutes. This patient was only slightly ataxic but was so frightened that he would scarcely trust himself to move alone. Total work done in fifteen minutes, 328 additions.

units of mental work, are done in each minute. The test continues for fifteen minutes.

After the test the total number of sums is estimated and the curve of the work from minute to minute is plotted on a sheet of ruled paper, by marking the minutes along the abscissa and by grading the ordinate according to the range of the number of sums per minute. On such a curve the initial spurt; the incidence, height and duration of the maximum working capacity; the time required for the attainment of the average working capacity; the onset and the rapidity of development of fatigue; lapses of attention; and the terminal spurt may be observed.

The results varied. Eight showed no detectable departure from normal. The others showed in varying degrees the efforts of fatigue and of diminished attention. The usual initial spurt was low and was quickly over, the maximum output was small, the work done from minute to minute varied considerably and tended to decrease steadily and the normal terminal spurt was not marked; the total output was subnormal. All of the investigated, except three, showed a considerable power of improvement; indeed, in eleven cases the improvement between the first and fifth tests exceeded two hundred per cent.

Because of this fatigue, attention is difficult to focus and quickly tires. This weakening of attention gives rise sometimes to sensory disturbances in tabetics similar to those seen in hysterics. Anesthesia may occur in bands or patches. If a ring be made round one of these anesthetic patches, the patch on subsequent testing may be found normal. More frequently the variability of attention is disclosed by pronounced oscillation in the sensory threshold at the same site at different periods. Similarly in testing vision, I have observed a tabetic one day give a majority of false responses when examined with a 32 candle-power light; another day give 84 per cent of correct answers when tested with a 4 candle-power light. If a slight resistance be opposed to abduction or adduction of the thigh, the tabetic sometimes either frankly ceases the attempt to move, or shows evidence in his facial expression of strenuous effort without any commensurate increase of force appearing in the movement. Indeed, palpation may detect no increased action in the muscle group which is being resisted. I have occasionally been able to support on one

finger the downward thrust of the heel of a seated muscular tabetic. Further emphasizing the functional nature of this muscular weakness, is the astounding gain in power which may be acquired by a short training in moving against resistance.

The mental stress of living with tabes varies from time to time according to changes in circumstances, favorably with rest and reassurance and in remissions of pains and crises, unfavorably in fatigue and in suffering. But during the course of the disease the average daily mental state tends gradually to deteriorate.

The rate of this deterioration is determined by the cerebral power which is itself dependent upon biological, environmental and nutritional conditions; by the extent and rapidity of development of the lesions; and by the conditions under which the tabetic lives. The chief of these conditions is the amount of mental strain to which he is daily subject. He finds he must exert greater and greater efforts to accomplish ordinary business and social tasks. His efforts may not suffice; his tasks may be badly done. If of the leisured classes, the tabetic gradually restricts his activities to his capacity. If the tabetic be an employee, he loses his situation, and, if impetuous, he drifts sooner or later to an almshouse. Usually his incapacity elicits from the tabetic less resentment than surrender. He does not, therefore, as a rule, develop a psychosis, but gives up the struggle and assumes a mental and physical immobility which is partly forced upon him but is mainly a defense reaction to his disabilities. This mental and physical immobility is seen to a greater or lesser extent in many of the hopeless, apathetic, bedridden tabetics, inmates of hospitals and institutions for chronic diseases. Tabetics in this

stage of parsimony of effort, of lack of initiative, of mental and physical disuse, are popularly but erroneously believed to be paralyzed. When the disease by incapacitating him renders daily stress less than his cerebral power, the mental state may remain stationary or may even improve—as in the blind.

The tabetic symptoms create the uncertainty which becomes fear, the stress which procures exhaustion, the hopelessness which leads to surrender.

The effect of the various symptoms upon the mental state varies not only with their intensity but also with their nature. Thus paresthesias may be less pernicious than pains or crises; double vision more disturbing than loss of taste or smell; ataxia more demoralizing than blindness.

Just as the symptoms react on the mental state, so the mental state reacts on the symptoms. In favorable mental states, pains, crises, blindness, deafness and ataxia are all diminished; in unfavorable mental states, increased. The attitude of the ataxic, his inappropriate wide base, his curious mixture of exaggerated and restricted movement, his rigid maintenance of the ill-judged muscular contractions which he thinks are indispensable to balancing, are largely an expression of the fear and tension in which he lives.

The influence of the mental state upon a symptom varies in different individuals according as their mental attitude. With equally extensive lesions the brave ataxic walks, the timorous is bedridden; a slight urinary disorder or a loss of sexual power may in one tabetic scarcely scratch the mental surface and in another produce intolerable distress; one bears cheerfully slight dullness of hearing, which in another becomes an isolat-

ing deafness. The mental influence varies also with the nature of the symptom. It predominates in motor symptoms, such as ataxia, because voluntary movement involves complex mental processes which are subject to every mental change. It is also of essential importance in a symptom such as pain, where the emotional is the chief attribute. But it little influences symptoms such as blindness and the feeling of walking on a thick yielding surface, symptoms which depend on a simple sensory perception and have a slight emotional content.

Symptoms are supposed to differ from signs in that the patient experiences the former, the physician discovers the latter; thus, pain is a symptom, an area of sensory loss is a sign. The mental state influences also the so-called signs. Tendon reflexes which on mental "reinforcement" increase, may temporarily disappear during the exhaustion which follows a bout of pain or a gastric crisis. The thresholds of perception of touch, pain, vision and deafness vary from moment to moment and from day to day, in correspondence with variations in the mental state. Postural sense loss and deafness may spontaneously improve in the blind. Indeed, the postural loss may be more a measure of the mental state than of the structural changes.

In estimating the reciprocal relations of the mental state and the tabetic symptoms, we may take as a guide any symptom. We may use the degree of pain, the variations in body weight, the frequency and severity of the crises, the amount of exertion necessary to induce fatigue, or other similarly variable factors. But the more objective the symptom the better index it affords. Ataxia, once it has developed, is the most evident, the easiest to measure and the most reliable guide, and it,

moreover, tends to displace pains and crises as an emotional expression.

In considering the ratio of the ataxic tendencies to the coördinating power, we saw (p. 78) that the lesions may be regarded as a constant, and the daily incentives to fear, anger, excitement, fatigue and depression, as a variable, in determining the liability to ataxia, the degree of ataxia: that ataxia, although partly a manifestation of structural change, is chiefly an emotional expression, a phenomenon of the mental state (p. 82); that the temporary aggravation of ataxia which accompanies blindness is due to the concomitant aggravation of the mental state: and that the subsequent, spontaneous disappearance of ataxia arises from the improvement which the enforced reduction of effort confers upon the mental state in the blind. We also saw that the length of the preataxic and of the ataxic periods was determined chiefly by the mental state. The mental state and this tabetic symptom are related as a mirrored image and its object.

The relationship between mental state and tabetic symptom implies the interdependence of the tabetic symptoms. Through the mental state a circuit is established between the various symptoms so that increase of one increases the others and reduction of one reduces the others. Aggravation of pain, or of crisis, or of bladder, or of other trouble, aggravates the mental state and increases ataxia; amelioration of any of these troubles improves the mental state and diminishes ataxia. Thus psychotherapy aimed at any symptom improves usually the others. The cure of ataxia by treatment or through blindness is accompanied by gain in weight, increased power to sleep, spontaneous diminution or cessation of pain, and often by returning virility. The ex-

tent of the improvement in coördination depends solely upon the amount to which the mental state improves.

Under any treatment in which one or more of his various disabilities are alleviated, or in which the patient has faith, the mental state improves. Hence medicinal substances with or without pharmacological worth gain repute. Boots and belts, if sufficiently lauded, produce more benefit than their mechanical qualities warrant. Electricity, baths, massage, and other measures produce results disproportionate to their merits. I have seen one ataxic who acquired marked benefit from Christian Science and another, from hypnosis. The influence of these measures depends to a greater or less degree upon the emotional appeal which they make. Thus, the influence of drugs upon the mental state is usually greater when they are administered by rubbing than by ingestion. The circumstance attending salvarsan injection, together with that drug's reputation, is responsible for much of the alleviation of pain, fatigue and depression, which sometimes follows its use; and this psychic effect is especially marked after the rubric which attends the intraspinal administration of salvarsanized serum. High frequency may be more potent than less esoteric forms of electricity, and electrical applications to the posterior urethra may be more beneficial than similar applications to atrophied muscles.

No skilful beginner can cause the mental state to improve as can the healer of popular repute. No method of treatment when first tried can influence the mental state so powerfully as when the faithful later flock to it for cure.

The value of any treatment of the mental state of tabetics is proved not by samples of its miraculous power

but by its ability to command success when administered by those who lack the personality of its protagonist and by its ability to heal those of little, as well as those of much, faith. Any method which every medical man can successfully use, and from which every tabetic shall derive benefit must be based upon:—

I. Knowledge of the mechanism of symptoms, and of the relative importance of the structural change and of the mental state in producing, in aggravating, and in perpetuating every symptom.

II. Knowledge of the reciprocal relations between symptoms and the mental state, and of the interdependence of symptoms.

III. Knowledge of the constitution, reactions, and capacity of the tabetic's psyche.

With this knowledge we can by treating the mental state, treat the symptom; and by treating the symptom, treat the mental state.

In treating the mental state, we avoid all circumstances detrimental to mental function, all occasions of fatigue, fear, depression or other unfavorable reaction. We procure mental rest and build up an available reserve of mental strength. We train attention and we teach order and system in mental action.

In treating the symptom, we avoid provoking it; we minimize it by mechanical means, such as braces, in ataxia; we palliate it by innocuous doses of drugs, such as phenacetin, antipyrin, and other so-called analgesics, in pain; we remedy it by physical therapy, such as massage, electricity and movement in muscular weakness; and so forth.

The cardinal treatment is the treatment expressly directed towards improving the mental state. When, as

in ataxia, the symptom reflects the mental state and the mental state is the chief source of the symptom, the treatment of the mental state and of the symptom are unified.

We shall consider first the general principles of treating the mental state, and then the auxiliary symptomatic treatment of the tabetic.

CHAPTER XI

TREATMENT OF THE MENTAL STATE: TREATMENT OF THE SYPHILITIC NEURASTHENIC AND OF THE PRE-ATAXIC TABETIC.

Imperative necessity for treatment of syphilitic neurasthenia, the precursor of tabes and paresis. Principles of treatment: rest, reduction of effort to capacity, avoidance of harmful thinking, training in mental control, freedom from unnecessary medical interference. Treatment of the preataxic tabetic. Prosecution for syphilis. Communicating the diagnosis. Reducing mental expenditure. Isolation and imprisonment. Immobilization and rest. Effect of attitude in perpetuating and aggravating stress. The teaching of rest. Rest exercises, respiratory and muscular. Psychological and physiological effect of the exercises; effect on blood pressure, pulse rate and pulse rhythm. Exercises in controlling mental processes through movement. Respiratory exercises, pneumograph. Head exercises, cephalograph. Eye exercises. Exercises in mental work. The reckoning test. The daily schedule. Results and duration of régime. The transition period. The return to business and social life. Systematizing and limiting daily effort. Importance of practicing the exercises to prevent relapses and to avoid ataxia.

ALTHOUGH a virulent spirochete may bring tabes to anyone, a non-virulent, we have stated (page 149), may make the nervous a tabetic or a paretic. As the nervous syphilitic is a potential tabetic, a potential general paretic, he, of all neurasthenics, imperatively requires unremitting care.

Syphilitics often complain of irritability; of the increasing readiness with which they experience fatigue; of tumultuous and irregular heart action; of lack of appetite, hyperacidity and nausea; and of constipation punctuated by diarrhea. These symptoms, common to states of mental stress, are due to increased tone of the vagus

and may be alleviated by a drug which reduces vagal tone, such as atropin.

But incomparably better than the palliative treatment of symptoms by drugs is the care of the mental state. The essence of that care is rest.

I lately treated a syphilitic in whom business tension due to the war caused a nervous breakdown, a cardiac neurosis with fear and sleeplessness. His devoted wife insisted upon nursing him: she quickly tired, and developed optic atrophy.

A necessary preliminary to rest is a careful examination of all of the neurasthenic's difficulties. The more thoroughly his problems and his reactions to them are investigated, the more successfully can he be aided. This investigation permits the reduction of the patient's effort to his capacity. It also affords opportunities to explain distressing reactions and to warn the neurasthenic against injurious thinking.

The neurasthenic should also be taught to control his mental processes, to think correctly. In such teaching, a beginning should be made with a task within his mental reach, a task which is part of his every day experience. It must require no new perception, no new mechanisms, no elusive interpretations. The mental processes involved in the task must have an outward equivalent by which their action may be judged, corrected, perfected. The mental processes which we can best externalize are those which have a measurable motor result.

Training of mental processes is, therefore, procured through training in movement. We desire a posture: its attempted attainment follows. We judge how the performance fulfills the desire. By repeated effort, we train ourselves to make the movement mirror the desire: the

act becomes effortless. From simple, complex movements are reached, all accurately performed and repeated in carefully regulated doses.

The effect of this training is not confined to the sphere of movement wherein it is gained. It influences for good all mental effort. It improves all mental activities. It inculcates the art of attending, of sustained effort, of logical sequence, of mental control.

The treatment of the syphilitic neurasthenic consists in these measures: it consists also in freedom from prosecution for syphilis: in freedom from depression by massive doses of mercury or arsenic; and in freedom from repeated examination of the cerebrospinal fluid. Heroic treatment, and over-zealous investigations, of non-essential attributes of syphilitic disease in the central nervous system, undoubtedly harm the neurasthenic. Variations in the cell-count, in the globulin content, and in the Wassermann reaction of the cerebrospinal fluid are often of less interest to him than to the serologist.

The beneficent effect of psychotherapy upon the course of the syphilitic invasion called tabes contains the promise that the intelligent care of the syphilitic neurasthenic may be powerful to retard that invasion.

The treatment of the preataxic tabetic does not differ in any essential particular from that of the syphilitic neurasthenic.

Some tabetics are ignorant of, but most suspect the syphilitic origin of their trouble. Experiencing the relentless consequences of a long past accident, the tabetic dreads the future not only for himself but for his family. In 1909, a young, married, preataxic tabetic, distressed for two years by a baffling pain in his legs, went with his father, a clergyman, to consult a knighted neu-

rologist of popular repute. After discovering absent knee-jerks, the knight impressively said to the tabetic, "I am now going to ask you a question which it would be equally my duty to put to the Archbishop of Canterbury, were he in your condition. Have you ever had syphilis?" The tabetic denied infection but was forced to admit, before his horrified father, that he had been exposed to the risk. The immediate scene and the subsequent estrangement of his family speedily resulted in severe ataxia, for which he was treated at the National Hospital, London, where I then was House Physician.

A discreet blood examination is more authoritative and more frank than the most minutely extracted confession. Other data regarding the infection and the family health may be obtained from the family physician or, later, by tactful questioning, without startling him or confirming his fears, from the patient himself. According to our present knowledge of tabes, the influence which most often makes the syphilitic develop tabes is mental stress. Mental stress results from overwork. Overwork is to a civilian as honorable a cause of unfitness as a bullet is to a soldier. And there is great mental ease in an honorable cause. The incrimination of overwork is not only humane, it is just; and it is the only basis of successful treatment of tabes.

There still attaches to tabes much of the pessimism of sixty years ago when German neurologists taught "*Ueber alle ist der Stab gebrochen.*" I have already mentioned my knowledge of two physicians, afflicted with tabes, committing suicide. A tabetic whom I have twice demonstrated, and who was formerly a medical student, six years ago consulted a doctor for intermittent pain in the stomach, accompanied by vomiting, and having no

relation to food. He next had transient double vision. Three years later sudden pains in his legs appeared, which another doctor called rheumatic. He visited a third doctor a year afterwards who, for the first time, associated the word "shooting" with the pain. There arose in the patient's memory a medical tag twenty years old, and he asked if "shooting pains" were not typical of locomotor ataxia. The doctor agreed that they were. "Have I locomotor ataxia?" "I don't know, I am no nerve specialist." The patient who had entered walking well, now greatly perturbed, with much difficulty reeled home. He was literally staggered by the suspicion that he had the much dreaded disease. Ataxia thereafter swiftly incapacitated him. His power to walk correctly had survived unimpaired through five years of his malady. A correct diagnosis was more pernicious to him in its immediate effects than five years of mistaken therapy.

When characteristic symptoms arise, too great tact cannot be shown in dealing with the incipient tabetic. The diagnosis may be communicated to the family physician: the last to hear it should be the patient or his family.

The first step in treating the preataxic tabetic is to reduce the expenditure of mental energy below the income. By progressively reducing effort, we may attain the level where a saving margin is daily attained. Usually, we institute a sudden rigid economy of effort by interrupting all social and business activity.

In reducing expenditure of mental energy, we do not merely isolate the patient with his worries. Prisoners in solitary confinement are more likely to go mad than to acquire tranquillity. Neither do we merely immobilize

him and call it rest. Many tabetics have been so rested who have never walked properly again.

To remove a patient from his problems is not to solve them. He carries their burden with him, a burden which the physician must at least share. Sympathetic attention, careful inquiry, experienced interpretation and advice, and, above all, systematic reassurance are necessary to bring ease to the preataxic tabetic. Even with such aid, given diligently, the tabetic in his pain and anxiety can often neither sleep nor rest adequately. He must be taught to rest.

According to James, we are ashamed because we blush, we fear because we tremble, we are pleased because we laugh. Without the blush we would feel no shame; without the trembling, no fear; without the laughter, no joy. Sherrington, after testing James' theory, concludes:

"In view of these general considerations and of the above experiments, we may with James accept visceral and organic sensations and the memories and associations of them as contributory to primitive emotions but we must regard them as reinforcing rather than irritating the psychosis. Organic and vascular reaction, though not the actual excitant of emotion, strengthens it. This is the kernel of the old contention about the actuality of emotion in the art of the artist. Hamlet's description of an artist as really moved by his expression may be accepted as an answer" (*The Integrative Action of the Nervous System*, pp. 267-268).

The attitude of the tabetic is an expression and a cause of the tension in which he lives. The attitude is muscular with cardiovascular and respiratory accompaniments. The removal of that expression relieves his mental tension. To remove it, I devised the following rest exercises:

The tabetic is instructed to breathe deeply and to pause at the end of inspiration and of expiration.

Having thus focussed his attention, he passes to breathing of moderate amplitude. The breathing is abdominal in type, slow in rate, and uniform in rhythm. The tabetic stops distracting thoughts as they arise, and de-

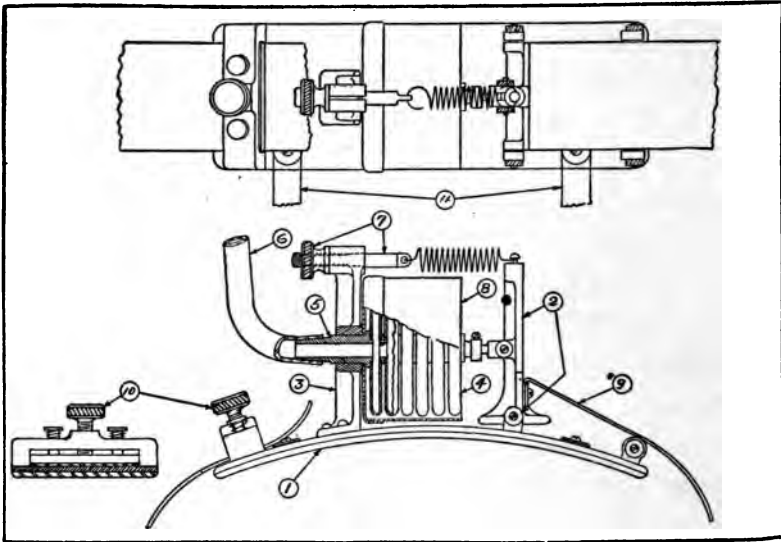


FIG. 70.—Description of parts as numbered on drawing of chest piece of pneumograph: 1, Breast plate lined with leather pad on concave surface. 2, Fulcrum plate showing mode of attachment to breast plate. 3, Fixation plate. 4, Diaphragm or metal tambour. 5, Outlet and hose connection to diaphragm. 6, Rubber hose connecting chest piece to recording apparatus. 7, Tension adjustment rod and nut. 8, Metal protecting cover over diaphragm. 9, Silk belt around chest. 10, Belt clamp for adjusting free end of silk belt to breast plate. 11, Ribbon suspending chest piece around the neck.

votes his attention solely to the sensations which accompany his breathing. A bag of sand or shot, or the physician's hand, placed upon the abdomen, increases the muscular effort so that the breathing may not easily lapse into an unconscious act. After moderate, gentle breathing is practised.

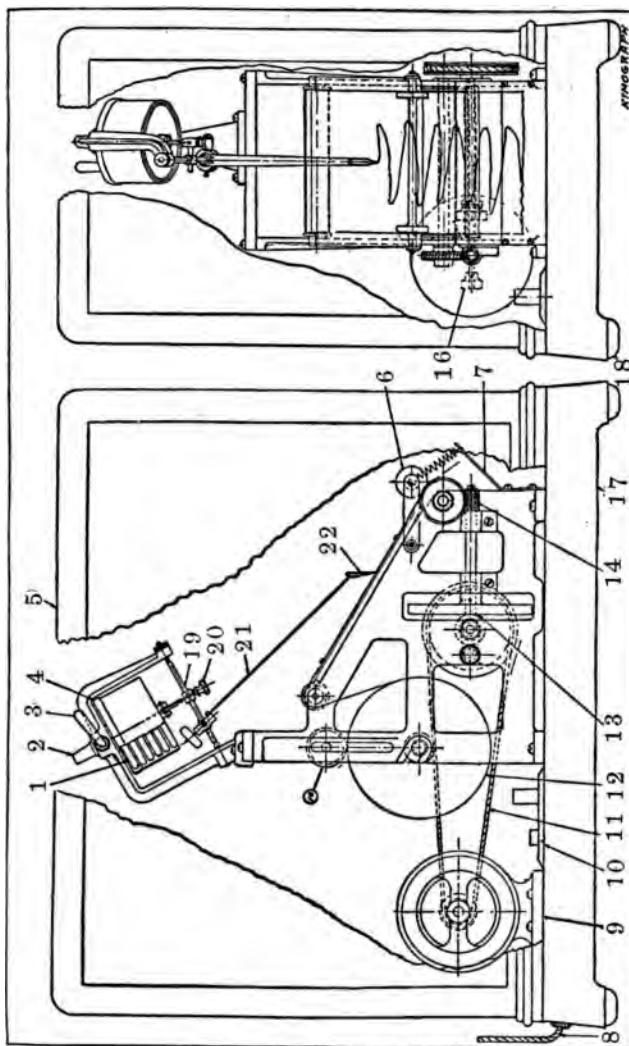


FIG. 71.—Parts as numbered on drawing of recording apparatus of Pneumograph: 1, Diaphragm or metal tambour. 2, Outlet and hose connection of diaphragm. 3, Air relief valve for adjusting tension of air in tambour system. 4, Metal protecting cover over diaphragm. 5, Glass case, to be removed when using apparatus. 6, Friction roll feed for unrolling paper. 7, Cut off plate for tearing off finished record. 8, Electric wire (plug on end connects to electric lamp socket). 9, Electric motor. 10, Push button switch for starting and stopping motor. 11, Steel wire coiled driving belt. 12, 75 yard roll of paper. 13, Friction drive for changing speed of paper from 4 to 24 inches per minute. 14, Worm and worm gear driving paper feed roll. 15, Push button for adjusting friction feed. 16, Paper weight roll for retard unrolling of paper. 17, Heavy iron base. 18, Rubber feet. 19, Pen movement. 20, Stroke adjustment screw for keeping pen arm moving in the center of paper. 21, Pen arm with pen point on lower end and balance weight on other end. 22, Glass pen to be filled with ink.

Then, muscular flaccidity is procured. Every limb is dealt with separately. Every joint is passively moved in turn. The muscles are first stretched slowly and steadily and next are similarly relaxed. When the prevailing muscular tension has thus been passively removed, the joints are free from muscular constraint; the

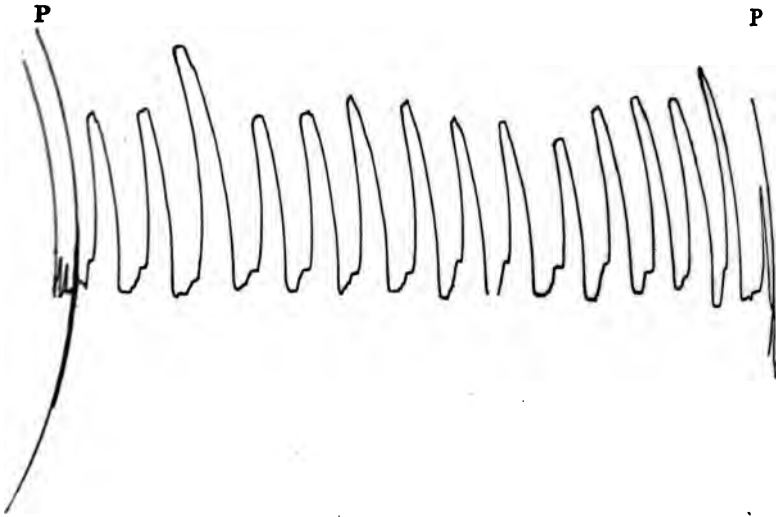


FIG. 72.—Up strokes represent inspiration, down strokes expiration.—Types of breathing disturbance. Note that inspiration, instead of being a continuous act, is performed in two or more parts. Expiration, the pause between inspiration and expiration, and as a rule, the pause between expiration and inspiration are here relatively unaffected. P indicates pause.

limbs lie flaccid and motionless; and fall limply from any unsupported position.

The scalp, forehead, cheek and jaw muscles of the recumbent patient are passively moved, till wrinkling diminishes or disappears, and muscular twitching ceases. After procuring flaccidity in the muscles of the first limb, the physician turns again to the scalp, forehead, cheeks, and jaw; relaxes those, and then again the limb. This linking of parts previously, to parts newly, freed from

muscular constraint, is helpful in bringing the whole to a satisfactory state of flaccidity.

The relaxation of the neck and trunk muscles is best accomplished while the patient sits. The patient's arms should then hang limply by his side, and his feet, crossed one over the other, should rest on their outer borders. The head should next be passively rotated in all directions, slowly and repeatedly. Then, by moving the supported trunk, the head is allowed to fall passively backward and forward, by its own weight. Finally, the head rests with the chin upon the chest, or hangs forward suspended by the ligaments of the neck.

The trunk should be slowly bent backward and forward and from side to side, until sufficient loosening of the

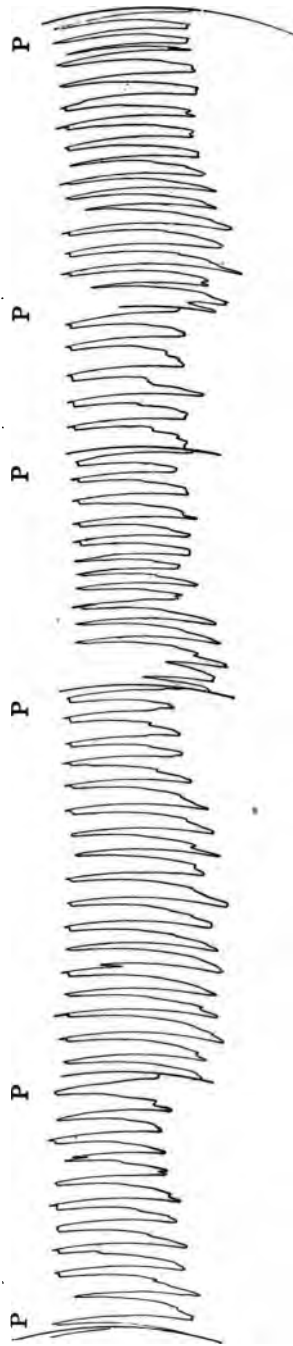


FIG. 73.—Types of breathing disturbances. Note the discontinuous nature of inspiration; the pause at the end of inspiration is almost invariably terminated by a short inspiration which in turn is immediately followed by expiration. At the end of expiration an analogous phenomenon is occasionally present. Sometimes inspiration begins, halts, and then begins again; or begins, is interrupted by a tiny expiration, and then proceeds once more. Expiration is usually much less disturbed than inspiration. P = pause.

muscles is obtained to allow the trunk to gravitate without restraint in whatever direction it is inclined, unsupported.

All movements should be passively made by the physi-



FIG. 74.—Types of breathing disturbances. Contrast the slowness and irregularity of inspiration, which is consciously performed, with the speed and uniformity of expiration, which occurs automatically. P indicates pause.

cian, and then, if possible, passively induced by the action of gravity.

Besides the tranquilizing of excited and anxious mental states and the removal of constraint from the muscu-



FIG. 75.—Patient A., Curve A.—Types of breathing disturbance. Jerkiness pervades the whole act, producing a staircase curve. The series of jerks and halts show that the muscles of respiration may produce chest movements analogous to the ataxic movements we observe in limbs. The following six curves show the gradual acquiring of respiratory control by the patient A.

lature, certain other physiological effects result from these exercises. A reduction of 25 to 30 mm. in high blood pressures commonly occurs. The reduction is not confined to high blood pressures: a fall of 10 to 15 mm.

happens in blood pressures which are nearly normal. As a rule, in normal and sometimes also in abnormal pressures, the fall affects the systolic and diastolic pressures equally; but in abnormally high pressures, the systolic usually falls more than the diastolic. In cases in which high blood pressure is not due to cardiorenal disease, the diastolic sometimes falls lower than the systolic. Indeed, in such cases, a fall is not invariable: a rise may occasionally occur, particularly in the diastolic pressure. Whether the blood pressure be lowered or raised by the exercises, the pulse pressure ratio, if abnormal, tends to approximate to normal.

Besides the fall of blood pressure, a decrease in the pulse rate, though sometimes slight, and rarely absent, as a rule is very evident. Sometimes the pulse rate falls thirty or forty beats a minute, usually ten to fifteen. The higher the initial pulse

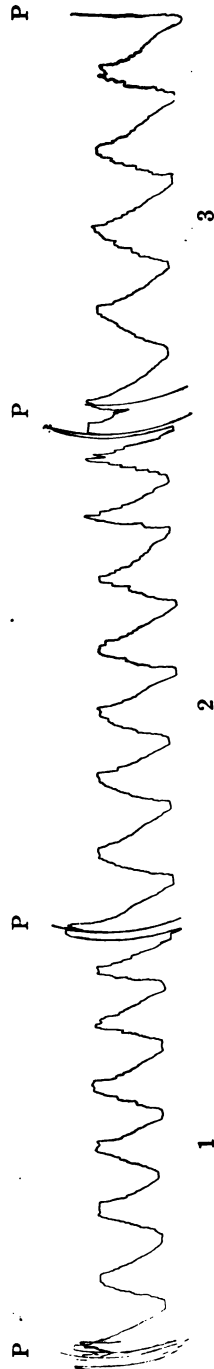


FIG. 76.—Curve A 2.—Practice in breathing begins with small movements and frequent rests. Note the exercises here were too long. (No. 1.) In the first exercise, after the third breath, respiration became markedly more irregular. This irregularity occurs even earlier in the second exercise, showing that the pause was not long enough. In the third exercise, after an inadequate pause, the patient breathes jerkily almost from the start. Under such circumstances longer pauses and shorter exercises are indicated. P = pause.

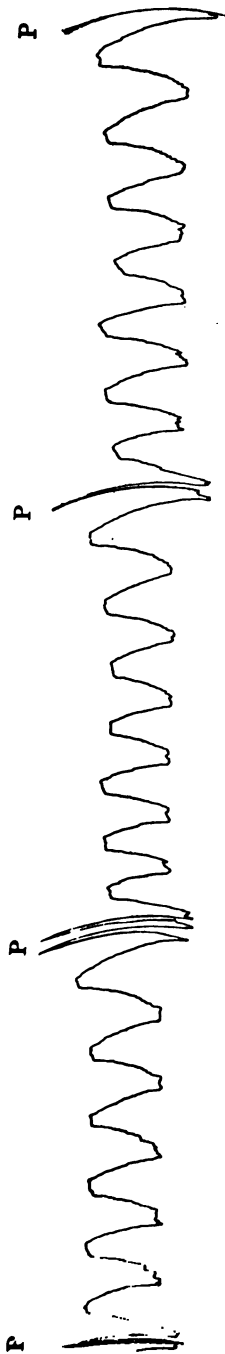


FIG. 77.—Curve A 3.—The pauses and exercises here are almost correctly adjusted. There is a tendency to jerkiness, especially between expiration and inspiration, but affecting somewhat the whole act of breathing. P = pause.

rate, the greater is the lowering. Besides the decrease in the pulse rate, pulse irregularities diminish or disappear during the exercises. Not only is rhythm restored but the pulse volume tends to become uniform.

Psychologically, these exercises produce concentration of attention. The physiological effects are relaxation of the voluntary muscles, diminution of muscular reflexes, slowing of the pulse rate, reestablishment of uniformity in the rhythm and volume of the pulse, lowering of blood pressure and restoration of the normal pulse-pressure ratio.

As a routine procedure, the tabetic is instructed simply and briefly in the essentials of the exercises. Then, in a quiet room, he lies on a couch sufficiently high to enable the physician to work without stooping unduly, and sufficiently broad to prevent the limbs from falling over the edge. The tabetic's head rests on a low and not too soft pillow. He is now told to breathe deeply. From deep breathing he passes to medium, and finally to gentle breathing. Then muscular relaxation is

systematically procured by passive movements, beginning with the face and limbs, and proceeding to the head and trunk. Throughout the passive movements, the physician periodically corrects the breathing, slows it as it quickens, insures adequate pauses at the end of inspiration and expiration, and encourages rhythmic, not staccato movements.

The objective guide to success is the behavior of the eyelids. When the lids are still, the pulse rate has slowed, and the blood pressure has fallen; when quivering returns, the tabetic is uncomfortable or distracted, and the pulse rate and the blood pressure have increased. By watching the eyelids, we can guess the progress, and we need not disturb the patient by frequent pulse or pressure observations.

The training usually begins with several ten minute exercises, separated by short intervals. As the tabetic learns, the exercises may be lengthened to three quarters of an hour. Five or ten lessons usually suffice to teach the whole procedure.

The easier he is to teach the less is the strain upon him, and the more pronounced is the relief of his muscular, vascular, and mental tension.

Most nervously exhausted tabetics have lost weight. They must, therefore, be fed with quantities sufficient not only to maintain life, to replace the daily cellular waste, but also to repair outstanding tissue destruction. It is the amount absorbed, not the amount ingested which counts. Meals should be frequent, ample, not excessive, daintily served, appetizing and easily digested. The elimination of waste products may need regulation.

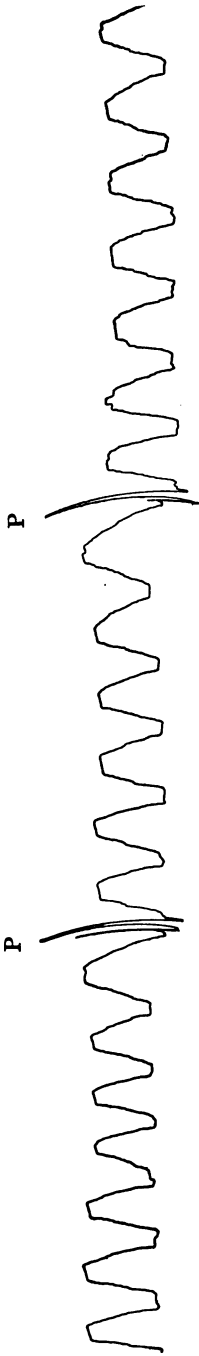


FIG. 78.—Curve A 4.—The breathing is becoming more steady. P = pause.

As useful adjuvants to stabilizing the vascular and, through it, the mental tension, cold baths are valuable. For those who react badly, first, cold sponging and drying of the body in sections; next, envelopment in a cold wet sheet accompanied by friction; then, high pressure douches for gradually lengthening periods at progressively lower temperatures, and finally alternating hot and cold douches, may be necessary as a training for cold baths.

The tabetic trained to rest is endowed with the necessary preliminary to all effort, the power to recuperate. Treatment must now be directed towards training his cerebral control. I begin with breathing exercises, for breathing is not arduous and it exerts a powerful influence not only over mental but also, as we have seen, over visceral functions. To use breathing as an exercise in control requires no apparatus. The method already outlined may suffice. But by graphically and visibly recording breathing by a pneumograph¹ such as Knauer and Maloney devised, the

¹Made by the Medical Machinery Company, Detroit.

respiratory movements may be immediately seen and any deviation from the normal, at once appreciated, checked, and corrected. Practice insures greater and greater control, more and more perfect breathing. At first at a slow, then at the normal, rate; at first sustained only for a short time, later for longer and longer periods; at first in quietness, and then with measured distraction, orderly breathing is attained. Later exercises comprise regularly recurring alterations in the duration of inspiration, of expiration and of the pauses between them, timed by the metronome. The mental state so far as it is reflected in the most delicate of all indices, respiration, becomes a matter of record, a source of reassurance and encouragement to both patient and physician.

To obviate swaying (Rombergism), to insure equilibration, and further to train the mental processes in ordered action, exercises in maintaining the head steady and in controlling head movements may be practised with the cephalograph. In every plane movements of regulated extent and rate may be performed. As the slightest movement is immediately recorded by the writing point, the patient can see any errors as

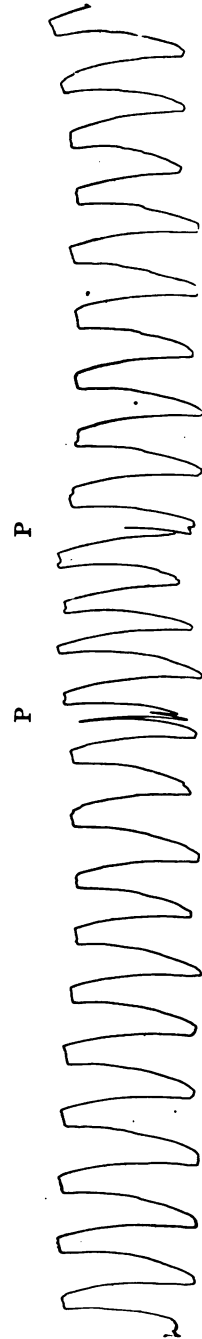


FIG. 79.—Curve A 5.—As breathing improves, deeper respirations and longer exercises are practised. P = pause.

they occur and may forthwith control and correct them.

Training in eye movements is also valuable. Every movement should be of prescribed direction, extent and rate. In such training a perimeter with a movable carriage may be used. The carriage bears a frame fixed by



FIG. 80.—Curve A 6.—Next, still deeper respirations are practised. P = pause.

a central pivot on which it can rotate. The frame holds two mirrors, the distance between which can be changed. Maintaining the head immovable, the patient sees the

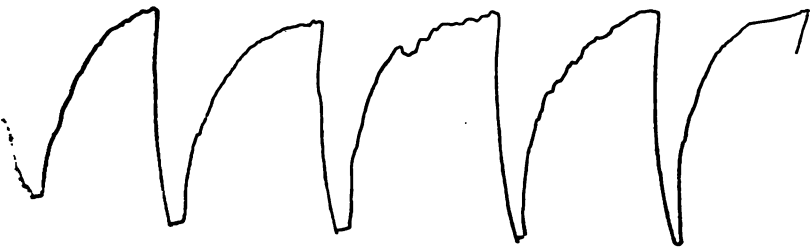


FIG. 51.—Curve A 7.—Then, slow deep breathing is taught. And the exercises are continued and varied until the patient acquires complete control over respiration.

image of, say, his right eye in a mirror at the fixation point. The mirror is then steadily moved in one direction for a certain distance, at a constant rate. So long as the patient looks in the mirror, his eye moves only in accordance with the movement of the mirror. If the

mirror moves beyond his range, he loses sight of the mirrored image. The eyes should be exercised in all directions; at first, singly, then conjointly; at first slowly, then more rapidly. Through the aid of such movements the squints and diplopia of tabes disappear in an astonishing manner.

Besides respiratory, head, and eye movements, other muscular exercises are of great value. These will be more fully described when we consider the treatment of the ataxic. All movements must be accurately defined, precisely performed, and carefully regulated in their time relations by means of the metronome.

The reckoning test is also of great value. I have already described (p. 180) its diagnostic use. Its employment periodically for a few days affords an objective criterion of the progress of a morbid mental state. But the reckoning test provides also a mental task the dosage of which can be graduated like that of any other therapeutic measure. Patients take a great interest in the test: the spirit of emulation is often astonishingly keen among them, and the knowledge of their daily results urges them to greater efforts. The therapy of psychic states cannot but welcome such a valuable accession to its strength.

The training in the rest exercises is itself the beginning of training in attention and in sustained and controlled effort. With the movement exercises or other tasks, a gentle beginning must be made. At the first attempt a few minutes' practice is enough. As soon as a pulse rises more than ten beats per minute, all effort should cease in favor of the rest exercises. Soon the time spent in work equals and then exceeds that spent in rest.

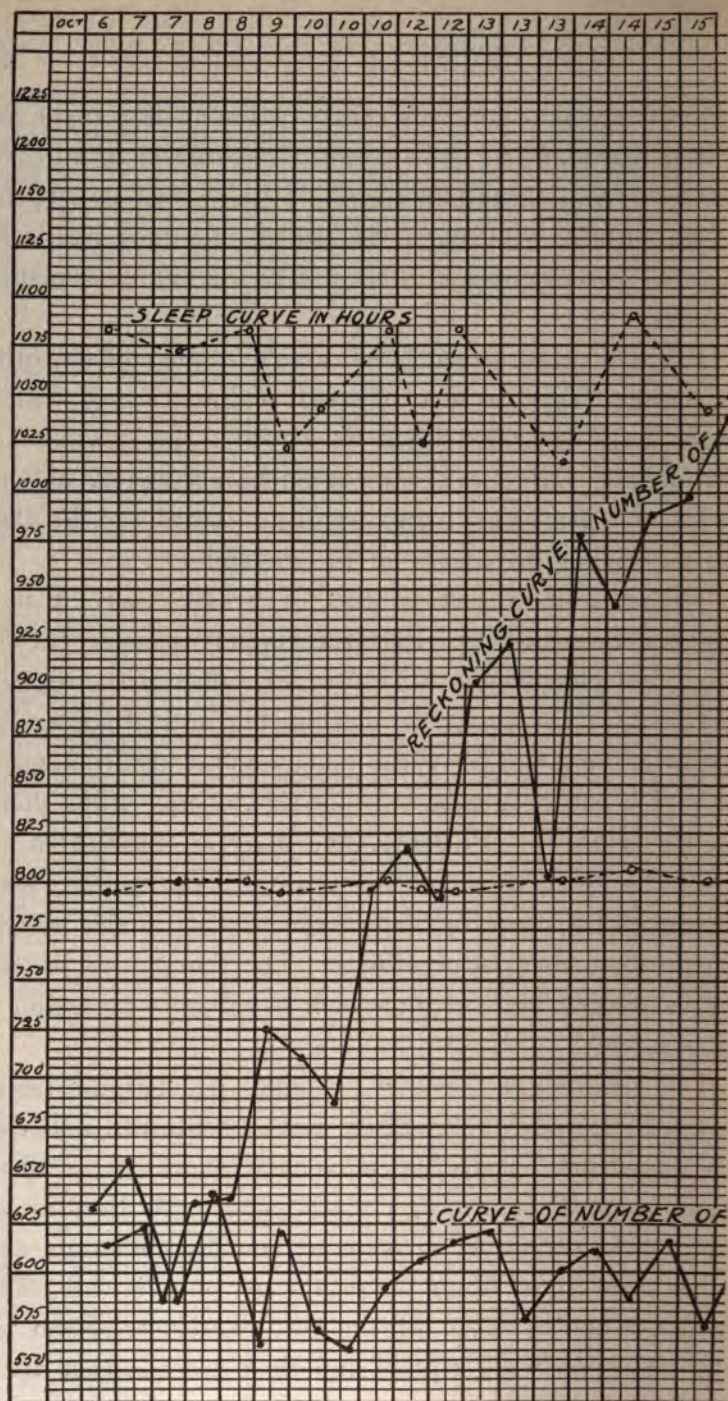
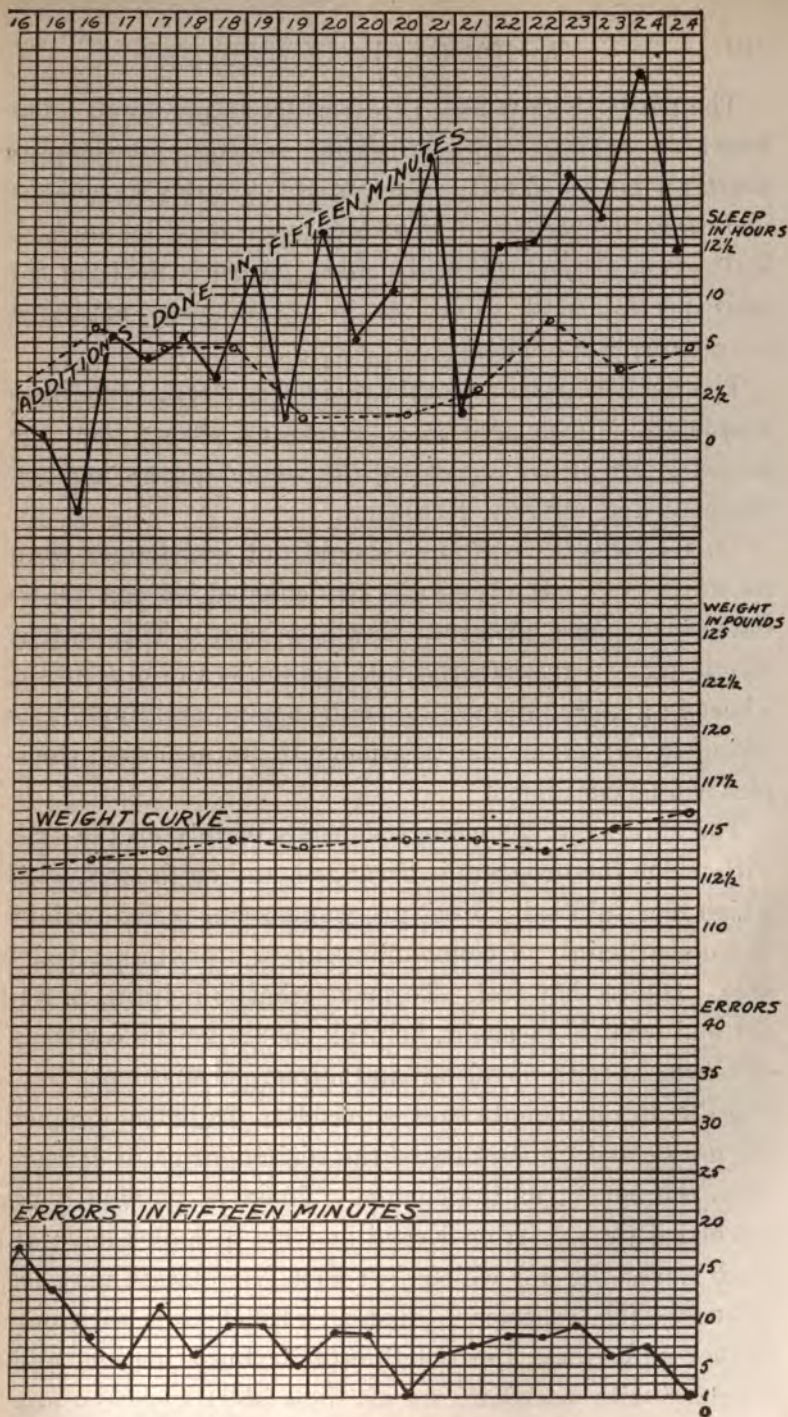


FIG. 82.—Chart of a patient beginning test, remarkable improvement in



treatment, showing by the reckoning
her capacity for simple mental tasks.

The patient's day must be carefully mapped out. The hours of waking, eating, working, resting and sleeping must all be scheduled. Each night the day's achievement must be recorded. The treatment must be methodical: every moment must be filled; there is no time for introspection and worrying in a properly arranged course of psychotherapy.

The earliest result of this régime is sleep, sometimes wonderfully prolonged and refreshing sleep; then the capacity for effort increases, the weight grows and excitement and anxiety decrease.

This intensive treatment should not last longer than its usefulness. If all results are charted we can ascertain the moment progress slows or halts. The patient should then be transferred with his nurse to the country, where in a more stimulating environment, and under less stringent direction, the treatment in its general principles can be continued.

The success of the change depends mainly on the skill with which the program is arranged, and on the tact with which it is fulfilled. Most programs seem to the patient to consist mainly of commandments to do nothing desirable. Hence they fail. To make what is helpful, pleasant and what is pleasant, helpful, should be the constant endeavor of both physician and nurse. Sometimes, fretting due to inadequate causes, baseless prejudice against the nurse, and intolerance of the régime, arise to detract from the benefit of the change. Those of us who have learned little else from Freud and his disciples have at least learnt the aid which just such complaints may be to successful treatment. Instead of listening with our former sufferance, the reaction of business instinct and courtesy with boredom, we are now alert to grasp any

clue to the patient's condition which may be revealed in his murmurings; and to persist in, or to change, details of treatment in a rational manner. The more direct the medical supervision the better can the régime be adapted and controlled. Hence, the tabetic should remain within reach of the physician.

Gradually, as isolation and discipline are now lessened, effort is increased, until the patient, having accumulated the necessary reserve of strength, and having gained in mental capacity, becomes fit to return to his former environment. Here the physician encounters usually his greatest difficulty—the adjustment of the tabetic's daily effort to his capacity. Obviously, to confront the patient again with the conditions which previously defeated him is to invite another disaster. But to suggest to a tabetic, not conscious of important physical deterioration, that he should give up his employment, alarms him and shakes his confidence. The adjustment must be made, but gradually, tactfully. So-called efficiency experts or system engineers may be an invaluable aid to redistributing the work of a business so as to relieve a tabetic proprietor of all the effort not needed for essential direction. An important business man may employ a secretary, allocate his routine work to his subordinates, start the day later, lengthen the midday recess, end earlier, and frequently use outdoor exercise, such as golf, as an excuse for a day's change. The commercial traveler, a frequent tabetic, may stop traveling and get an indoor post; the telephone inspector may change from outdoor to office duty; the naval commander may leave an arduous position on shore for a peaceful mission on the high seas; the laborer may labor mildly on piece work or become a janitor.

Whatever change is made should be timely and should be introduced as a strictly temporary measure. If the change be adequate, if no further signs develop, greater activity may gradually be resumed. Fluctuations occur; their importance must not be overestimated; the average condition as regards sleep, fatiguability and weight from week to week is the chief guide to the success of the retrenchment of effort.

The initial effect of reorganizing the daily effort may not be reassuring. The tabetic may fret and may have difficulty in restricting his endeavors to their new scope. If the effort demanded in the new task is not too great, this stage of dissatisfaction will soon pass. But the change may be really unsuitable. Thus my naval commander's peaceful trip was unexpectedly ended by the menace of war and he had to resign; the inspector finding office routine much more arduous than previous experience had led him to expect, took up other work: one has now been a preataxic tabetic for seven years, the other for six years.

In addition to restricting effort, the tabetic must use the rest exercises assiduously. He is particularly exhorted to rest after any strenuous or exciting occurrence. When the stress of circumstances in daily life causes nervous tension, he must not permit it to endure. Together with the shortening of periods of high tension and the maintenance of a lower average level, these exercises, systematically practiced, cause oscillations in tension to be less easily induced, less frequent, and of smaller extent. The tabetic should also continue to practice his exercises in movements of regular rate, graduated duration and accurate extent. These movements should be performed with the utmost attention to every

detail, in order to regulate and strengthen his control over psychic processes and to prevent the usurpation of cerebral power by unnecessary and tyrannical symptoms, such as ataxia.

CHAPTER XII

PSYCHOTHERAPY OF PRETAXIC SYMPTOMS

Pretaxic symptoms. Diplopia. Pain, measurement and analysis. Treatment of subacute pain; of pain crises. Gastric crises, measurement, prevention. Treatment of gastric crises. Cocain. Morphin. Root section. General principles of treating pain, gastric, and other crises. Involuntary micturition; prevention, treatment. Intestinal and genital symptoms. Blindness. Deafness.

REDUCING the expenditure of mental energy below the income, and training cerebral action so that achievement may be commensurate with effort are, we have seen, the essential principles upon which we base the treatment of the syphilitic neurasthenic, the potential parietic, the potential tabetic. During the incipient stage of tabes, the neuralgic, or pretaxic stage, the only stage which should ever be seen in tabes, these principles are rigorously applied. As the tabetic symptom and the mental state are reciprocally related, this treatment of the mental state prevents or minimizes the tabetic symptoms.

Unfortunately, we seldom have the opportunity to prevent symptoms. Patients seek our aid only when symptoms compel recourse to us. Then, to give relief from the obnoxious symptom, we treat the symptom mainly through the mental state or the mental state mainly through the symptom, according to the nature of the symptom (p. 185). The treatment of mental state and symptom are complementary.

Before beginning the treatment of any symptom, we must, if possible, measure it. Measurement is the only

means by which we can accurately estimate the results of treatment.

After measuring the symptom as precisely as possible, we endeavor from a study of the extent of the lesions and of the mental state, to determine the importance of the structural and of the mental changes in the production of the symptom. The less the lesions are incriminated, the more the mental state is blamable, and the more rapidly and successfully will the symptom yield to treatment.

During the preataxic period, the symptoms for which relief are sought are "nervousness," weakness and fatigue, diplopia, pain, crises, bladder trouble, blindness and deafness.

The treatment already outlined will quickly relieve nervousness and fatigue.

Diplopia may be investigated and measured by the methods previously described (p. 107). It usually spontaneously disappears. It seldom persists more than a few days after exercises in eye movements (p. 206) are begun. By such movements squint may often be removed, even when of long duration.

The benefits conferred by oculists through treatment of refraction errors emphasize the importance of diplopia and of visual troubles as causal factors in the mental state of the tabetic. Patients dislike to wear a patch over the misleading eye. I insist on the patch only when conscious diplopia is present. Attempts to remove the need for unconscious suppression of the false image by correlating the eye movements are usually partially, but are seldom completely, successful.

Few symptoms have a larger mental element than has pain. By pain the tabetic accounts to himself for his

feeling of incapacity; by pain he excuses his lack of effort. It is at once his plea, his sentence and his pardon. One of my tabetics, who for twelve years conserved his pain without detriment to his physique, habitually slept undisturbed through the night; awoke painlessly towards 7 A. M.; arose and began to dress. At this stage in his daily routine he was seized with pain, which he expressed by crying and by gyrating about his bedroom. Pain and this mental state were practically his only symptoms. The hour of its onset suggests that the pain was provoked by a more or less unconscious dread of facing the day's work. When I first saw him he had maintained this curious matutinal reaction for years, during which his able and devoted wife had successfully assumed the burden of earning for both.

One suffers lightly a pain which another finds words inadequate to describe. A chronic pain which the tabetic bewails, which permits sleep of ordinary duration, and which is unaccompanied by loss of weight, has not the importance which the tabetic gives it. The same lesions that produce obsessing anguish in one may cause in another only an insignificant ache. The immensity of the pain in the first is as real as its triviality in the second. The respiratory movements afford a most delicate index to mental processes. We can roughly measure and record the mental reaction to pain by applying stimuli to the tabetic while his respiratory curve is being graphically recorded.

Subjective pain in the tabetic is thus of two kinds; one, mainly structural in origin, intermittent, transient, and tending spontaneously to cease. The other pain probably once had a structural basis but it now persists chiefly as a phenomenon of the tabetic's mental state.

This latter pain, with its pseudo-crises, waxes and wanes. It is greater in unfavorable, less in favorable, mental states. Its place tends later to be usurped by ataxia which affords the tabetic a more adequate expression of his mental reactions. Hence, when ataxia develops, pain often decreases. This type is very amenable to psychotherapy.

I never inquire about pain unless the tabetic reports it. If it be troublesome he will surely complain. The testing of the mental reaction to pain by the effect upon respiration of pinpricks and other slightly painful stimuli; and consideration of the patient's weight, sleeping and working capacity, together with his mental state, enable the physician to form a fairly just opinion of the nature and real importance of this symptom. Lack of appreciation of the true basis of the pain state leads to futile alcohol injections and to posterior root sections in tabetics who would suffer pain if they possessed not a single surviving pain fiber.

Pain is an ideal symptom to treat by indirection, to treat through the mental state. The avoidance of all circumstances unfavorable to mental action, the insuring of rest and the regulation of the mental processes alleviate, and often banish, it. Pain must not be allowed to interfere with treatment. No matter how much complaint be made, the rest and movement exercises should be attempted. If the effort be too great for the patient, his pulse will increase in rate, his respiration will become jerky and other objective signs of failure will be evident. Usually after a few minutes' exercising, complaints cease, and the end of the session finds the patient completely relieved. The exercises are the most logical and effective method of treatment we possess for pain. As

the mental state improves, pains diminish in frequency and severity, till the tabetic ceases to mention them.

I seldom use analgesics for subacute pain. The rest exercises and local heat usually enable the patient to command relief. Occasionally, high frequency has been useful to reinforce other mental influences. Adequate bracing of the foot seems also to exert a benign influence on pain.

Crises of pain are mercifully transient and tend spontaneously to disappear. In crises, every palliative except alcohol and opium and the latter's derivatives may be tried. Phenazonum, five grains, acetanilid, three grains, and caffen, two grains, constitute the powder I use. In very severe cases, an injection of $\frac{1}{16}$ gr. of hyoscin hydrobromid may be serviceable.

There are cases reported of pain enduring thirty years and being the unique source of complaint in a tabetic. We are grateful if we can so far protect the tabetic that pain, which does not materially diminish his capacity, is the sole manifestation of his disease. Such pain is an emotional outlet and is invaluable to him as a warning and a curb when he attempts to transgress the limits of safety in effort.

Gastric crises are much more satisfactory to treat than pain. We can see a gastric crisis and from the amount, nature, and frequency of the vomiting, and from the patient's weight, we can estimate its severity. But as the attacks are self-limiting, as they do not recur for months, and sometimes even for years, as in some tabetics they are frequent, in others rare, and in others, again, unknown, we cannot at present with certainty decide the efficacy of treatment. But if we study the cause of the vagal explosion which constitutes a gastric crisis (p.

128), and treat it rationally, we may share with the tabetic his satisfaction from the alleviation which occurs, even if we cannot substantiate our claim to the credit.

I have already dealt with the condition of vagotonia (p. 190) which manifests itself in gastric discomfort, hyperacidity, nausea and vomiting; which is due to mental stress, and which may be a prelude to gastric crises. The palliative treatment of these incipient crises we saw consisted in reducing vagal activity by atropin, hyoscin, and allied drugs which paralyze the vagus. To insure speedy action, accurate dosing and psychological effect, and also to avoid possible gastric disturbance from ingestion, these drugs should be administered by subcutaneous injection. Their power may be usefully reinforced through stimulation of the sympathetic by adrenalin administered per rectum. The minimizing of local irritation by careful dieting should not be neglected.

But the essential, the curative, treatment consists in improving that mental state to which the vagotonia is attributable. In the study of the mental state which precedes its treatment, special attention should be directed to the possible existence of direct incentives to nausea, of complexes, especially fear complexes, which utilize nausea as an emotional expression.

During the attack, rest is the sovereign aid. This rest must be mental; the patient should be kept isolated, quiet and reassured: and physical; the patient should lie flat on his back in bed without pillows: and local; no food or drink or drug should add to the gastric irritability. An ice bag applied to the epigastrium is sometimes valuable.

In addition to these measures, innocuous doses of local and general sedating drugs are used. The local drug

most lauded is cocain. It is supposed to anesthetize the gastric mucous membrane. The amounts recommended must be without appreciable effect upon the large area involved. Any anesthesia which cocain induces lasts only minutes. The subsequent irritative reaction to it is prolonged. Its use for gastric crises should be discountenanced.

Of general drugs, morphin and its derivatives are most used. The speedy and complete oblivion to his troubles which the tabetic derives from morphin endears this drug to him. Morphin weakens him mentally and physically. When once used, it demands repetition. It becomes a reward for crises. It brings local and general infections in its train. There is no more lethal agent to the tabetic than morphin.

Gastric crises seldom last more than forty-eight hours. Easily assimilated food, at first fluid, later mixed, then solid; at first, in small measured quantities, later in larger; at first, at infrequent, later at frequent intervals, soon restores to the tabetic any weight he may have lost.

I have luckily never encountered a case in which life was menaced by gastric crises. But by those who have, heroic treatment has been advocated. Förster recommended the cutting of certain posterior roots, to sever some of the sensory fibers which proceed from the stomach to the central nervous system. If such root section can prevent gastric crises, then an intraspinal injection of novocain at the level of these roots should be able abruptly to terminate the crisis. I am not aware of any satisfactory demonstration of this power. I tried it twice without enough success to tempt me to try it again. I have no personal experience of Förster's operation. The only case in which it was practiced, within my

immediate circle, died. Of twenty-five resections of the seventh to the ninth or tenth posterior thoracic roots, for gastric crises, reported by Förster to the London Royal Society of Medicine, in 1911, three resulted in death, two failed, and twenty were called successful. In only thirteen of these twenty was there no return of the crises; only two of these thirteen had been observed for eighteen months. There is, therefore, a mortality in the hands of the protagonist of this treatment of twelve per cent. For this mortality, the maximum success demonstrated is relief from crises for eighteen months in two out of twenty-five cases, i.e., in eight per cent. Such success is dearly bought. Ere now there have probably occurred more deaths from this operation alone than are attributed to gastric crises in all medical literature since tabes was first recognized. The failure of the operation has only led Förster to recommend the extension of the operative field, and Exner to advise the section of the tenth, eleventh and twelfth roots.

Root section has also been tried for the relief of lightning pains; but it has fortunately been abandoned.

When a pain, a bladder, a rectal, a gastric, a laryngeal or other crisis appears, it should be palliated; when it passes, its effects should be repaired; provocation of a recurrence should be avoided; and the mental state should be zealously treated. The success of psychotherapy and the infrequency with which pain or gastric or other crises interrupt the placidity of the blind, show that alleviating the mental state is, at present, the best means at our disposal for treating these symptoms.

Excepting diplopia, involuntary micturition is the easiest of the tabetic symptoms to cure. Involuntary micturition, as I have pointed out (p. 134), is due to the

tabetic's habit of infrequently and imperfectly emptying his bladder. It is a symptom which should never occur. Every tabetic should be warned that he must urinate at definite hours irrespective of his need. If a tabetic under medical treatment develops dribbling, it implies either his disobedience or the physician's neglect.

In bladder disturbance complicated by cystitis, the bladder should be mechanically emptied at regular and frequent intervals and should be flushed by bland warm saline solutions till the returning fluid is clear. For this purpose only a sterile soft rubber catheter is permissible.

Usually all that is needed is urination at prescribed hours. In urinating, the incontinent tabetic should assume the attitude which is mechanically most favorable for emptying the bladder, the position in which the intra-abdominal pressure is highest, the crouching, squatting attitude, a low sitting attitude in which the thighs are well flexed on the abdomen.

The aid of the sound of running water may be helpful. Urination should be attempted for five minutes every two hours. No fluid should be taken for two hours before retiring; and at first, the bladder may then need to be emptied by catheter.

This regular emptying, without waiting for sensory demands, prevents accumulation of urine in the bladder, and maintains the bulk of the bladder contents below the containing capacity. Dribbling is therefore avoided and with it the distressing soiling of the tabetic's garments. The bladder wall is no longer unduly distended. The stretched blood vessels relax and dilate. The blood supply increases and the bladder muscle and sphincter regain their tone. With increase of tone, the containing power of the bladder and the expelling force grow.

During urination the patient should practice breathing and relaxation exercises and should attend to the sensations which accompany the urinary act. This attention enables him to perceive the feeble, imperfect sensory images which he has been ignoring; and soon he acquires dependence upon them.

The urinary intervals should gradually be lengthened, the attitude should progressively approach that normally employed. Usually only two or three weeks' practice is needed to reestablish a useful degree of control.

Analogous measures relieve difficulty in defecation. I have already mentioned the return of virility which patients whose mental state is being successfully treated not uncommonly report.

In tabetic optic atrophy, the measurable variation of visual acuity which accompanies alteration in the mental state seems to promise that psychotherapy will benignly influence blindness. But I have never succeeded in producing in tabetics who are becoming blind a mental state which was stable and permanently favorable to vision. The impending darkness daunts them beyond reassurance. Nor have I, in the few cases in which I have tried, been able to arrest the progress by complete visual rest and by psychotherapy. In two cases who were rapidly losing their sight, as a last resort, I used mescaline (p. 88), hoping I might restore their confidence and conserve some of the drug's action, by demonstrating to them that they possessed a visual power so far exceeding what they daily used. In neither did the increased acuity survive the action of the drug. The only cases in which I have been able to benefit vision to a measurable extent by psychotherapy are those in whom merely perception of light remained. If the syphilitic invasion finally halts

before this stage is reached, analogous improvement should be procurable; but I have no experience of it. Treatment of the mental state of the blind tabetic renders his blindness less intolerable. The measures I have outlined are for the blind tabetic merely a prelude to useful occupational exercises.

The auditory capacity in the seemingly deaf is often astounding. Unsuspected by the physician even a sufficient number of cochlear fibers may persist to allow the reestablishment of a useful degree of hearing.

During the improvement in the mental state which accompanies rest and exercises, hearing spontaneously improves. The recovery of hearing sometimes takes an unusual course which further emphasizes its psychic nature; thus, one of my tabetics, deaf as the proverbial post in ordinary social intercourse, now experiences no difficulty in hearing telephonic communications. Apart from training the mental state and teaching the importance of hearing the first word—that is, of immediately directing all attention to hearing—I have not attempted to treat the deaf tabetic.

In a paper which I read to the Otological Section of the New York Academy of Medicine, in 1914, I emphasized the psychic factor in deafness and the tendency of deafness to increase with stationary lesions. I pointed out that there is a more or less dominant tone in every sound by which we distinguish and interpret it; and that in the same sound we each may recognize a different tone as dominant. I suggested that those trained to recognize a sound by one tone may, if the fibers conducting that tone be destroyed, learn to recognize it by some other tone subserved by fibers which have escaped. I, therefore, urged that every deaf tabetic should be carefully

tested by means of the Edelman-Bezold, or a kindred apparatus, throughout the entire range of the human auditory scale; that the surviving tones should be carefully charted; that training in the perception of the surviving tones, at first, tone by tone, in varying intensities, and next in combination should then be practiced; and that, finally, the interpretation of sounds by means of these tones should be taught. Blindfolding will make this training easier and more successful.

CHAPTER XIII

TREATMENT OF MOTOR SYMPTOMS OF TABES

Faulty equilibration first motor disorder in tabes: its measurement, analysis, and prognosis. Ataxia, its prevention and measurement. Minimizing ataxia by reducing movement and by mechanical supports. Defects of the tabetic foot as a support. Means of ascertaining and of remedying these defects. New method of obtaining cast of foot in action. Foot brace. Special tabetic shoe. Braces for joints. Value of belt and bandages. Temporary use of all appliances.

ROMBERG'S sign, swaying abnormally when standing with closed eyes, is the first indication in the tabetic of inadequate mental control over movement.

The amount of swaying may be measured by the cephalograph which records not only the extent but also the planes of the swaying. But this instrument is inessential. We can roughly estimate the degree to which equilibration is disturbed by the attitude necessary to make its disturbance apparent; or by the attitude necessary to insure stability; or by the length of time the highest stable attitude can be maintained.

In addition to measuring the power to maintain the attitude of the body as a whole, we measure the power to maintain the position of every limb, and of every section of a limb, and the postural sense in the respective joints.

The primary cause of the deterioration of attitude, the underlying root or vestibular lesions, should be sought through the motor and sensory signs which reveal them. The less extensive the lesions are, the greater is the

disparity between the loss of postural sense in the distal and in the proximal joints; and between the loss of postural sense and the loss of the power to maintain posture. With this disparity will be found evidence of mental fatigue, of lack of attention, and of rapid development of the instability—all signs of a dominant mental element in the symptom, all promises that speedy and gratifying improvement will follow appropriate mental treatment.

If the mental treatment of the pretabetic be timely and adequate, lack of equilibration will seldom develop. If lack of equilibration be promptly and wisely treated, ataxia will not occur, except as the direct consequence of some sudden calamity. As the treatment of instability differs in no essential from the treatment of ataxia, and as, in practice, instability and ataxia usually together confront us, to avoid repetition, we shall deal with the treatment of instability as it arises in the course of ataxia.

We saw (p. 77) that all the influences which conduce to fear, fatigue, depression, excitement, anger and other mental states unfavorable to movement constitute so many incentives to ataxia, so many ataxic complexes; and that the liability to ataxia, or the measure of ataxia, may be expressed by the ratio:

$$\text{Coefficient of ataxia} = \frac{\text{Ataxic complexes.}}{\text{Coördinating power.}}$$

When the value of this coefficient equals unity the threshold of ataxia is almost reached.

In treating the preataxic tabetic, our aims were to reduce his effort to his capacity; to remove influences which perpetuated his mental stress; to train him in rest, to enable him to accumulate a reserve of mental energy;

and finally to train him in cerebral control so as to make the results he attained commensurate with his efforts. The achievement of these aims would reduce the ataxic complexes, the numerator of this ratio, and increase the cerebral control, the denominator; would lessen, therefore, the value of the coefficient of ataxia, would prevent ataxia. The tabetic who is ataxic is merely a tabetic whose mental state has been allowed to deteriorate below the level necessary for effective action, a tabetic whose treatment has been neglected.

Before treating an ataxic, we determine by analysis of his attitude and movement the essential factors in their disorder and we obtain a record of the ataxia.

In order to record his gait, the ataxic dips his feet, clad in socks, into a solution of potassium permanganate. If he can walk only with the aid of a stick, the end of his stick is covered with cotton fixed by thread and also dipped in the permanganate. He then walks upon a strip of paper, 40 inches wide. This paper is that used for the printing of newspapers, and is supplied in a large roll. Each patient walks about fifteen paces, and, as he walks, the imprints of the end of his stick and of his feet are left upon the paper. This imprinted paper is then cut off, dated, and the name written upon it. For purposes of reproduction, it is photographed on a greatly reduced scale (*see* Figure 83). Subsequent sets of foot impres-



Fig. 83.—Chart of gait in early case of locomotor ataxia. Reduced to one-ninetieth. Chart cut in sections to facilitate reproduction.

sions graphically record the progress of the treatment.

The mental state is woefully deranged by the onset of this inexplicable, bewildering, relentless symptom, ataxia, which threatens the very essence of activity, movement. And every movement, according to the degree to which it is ataxic, further decomposes the mental state.

The more an ataxic walks, the more ataxic he becomes. It is difficult to cure a symptom which is constantly being aggravated. No mental relief is so great as that which arises from the relief of the ataxia. The most obvious relief measure is reducing movement—the use of conveyances, the bringing of the home nearer to the place of work or business. But still more valuable are mechanical aids to stability. Any ataxic who can walk with a stick can walk without a stick. Yet the stick immediately reduces the effort to walk. The more advanced the case, the greater is the benefit which mechanical aid confers. The kind and the amount of the needed aid differ in almost every case, and for any particular case can be ascertained only by careful examination. To inflict unnecessary appliances is as unwise as to deny needed support. I shall describe the mechanical treatment of a case of ataxia of average severity, merely as an index of the mode in which the problem should be confronted.

When standing, the average ataxic tabetic rotates his feet outwards in a horizontal plane, around a vertical axis, which passes through the ankle joint, to such an extent that the distance between the great toes may be several times that which separates the heels. These abducted feet are sometimes overpronated, and in such cases both the anteroposterior and transverse arches

usually yield. Such a foot affords a wretchedly inadequate support. The only stable element in it may be the os calcis: the foot may move as a flail attached to the ankle: in front of the os calcis, it may be only



FIG. 84.—Footprints showing the habitual attitude of the feet in the slightly ataxic. Note the distance between the heels is much less than that between the big toes.

a flabby conglomeration of bones. The hypotonic muscles of such a foot elongate. The ligaments stretch perhaps beyond functional utility. I have seen an increase of an inch in length and of an inch in breadth occur when the body weight was placed upon the foot. The tabetic with these abducted, pronated, formless feet, in his necessity, clings to the ground with his toes. During this simian action, which is partly a reflex expression of fear, the middle joints of the toes are arched against the box of the shoe. The ataxic, moreover, often dorsiflexes his foot and toes as a prelude

to the movement of flexion at the hip joint in walking

Sometimes the abducted feet are supinated, the patient walking on the os calcis and base of the fifth metatarsal. In such feet the clutching with the toes is even more marked than in the pronated feet; but although both arches suffer, the displacement of the tarsal bones is less constant.

The clutching of the ground with the toes and the excessive dorsiflexion of the foot cause blisters and callous formation on the tips of the toes, where they rub against the sole of the shoe, and over the dorsal aspect of the middle joints, especially of the second and third toes, which come in contact with the box of the shoe.

The lack of support which the feet afford to the swaying tabetic creates in him not merely uncertainty at every step, but even a lively fear. The instability and fear result in prodigal expenditure of muscular and nervous energy and contribute to fatigue. Thus, the troubles of the ambulant ataxic multiply.

When the feet are separated so that each external malleolus is in the same vertical plane as the great trochanter, and the long axis of each foot is directed forward, making the feet parallel (*see* Fig. 94), the feet are in the position which confers the greatest amount of stability in standing. In this position, the weight, bearing on each foot, is distributed across the longitudinal and transverse arches, and is directly transmitted to the ground by a tripod, consisting first, of the internal tubercle of the os calcis; second, of the sesamoid bones and the head of the first metatarsal; and third, of the base of the fifth metatarsal.



FIG. 85.—Shows initial state of a tabetic. The blurring of the picture is due to his unsteadiness. Note the broad base, contorted attitude, and the need of the stick in standing. A second photograph (Fig. 94) was taken six weeks later. (Grossman.)

The tabetic foot should be braced in such a manner that in this proper position the full weight will be borne on the natural tripod in standing and in walking. In order to achieve this accurately, in addition to the usual



FIG. 86.—Shows broad base, crouching attitude and the anxious vigilance necessary to maintain it.

procedure of recognizing a diminution in the longitudinal arch, and correcting this along the lines indicated by a cast of the foot taken in a position of rest, we need to obtain a cast of the foot in the act of bearing the full weight of the body. A comparison of this cast with that taken of the foot at rest shows exactly the points on which the weight is falling, the degree to which the natural arches have given way, and the amount of spread in all directions, which is permitted by the overstretching of the ligaments and muscles.

To obtain the cast of the foot in action, Dr. Sora-

pure and I proceed as follows: Two tin-plate boxes, fourteen inches long and seven inches wide, with removable sides two inches high, are half filled with dental wax, which has a melting point of 110° F. The boxes accommodate any size of foot. They can be fixed parallel to one



FIG. 87.—Footprints of tabetics. As all possible variations in the longitudinal arch may thus be encountered, there is no standard brace for the tabetic foot; what helps one patient may injure another; every tabetic foot should be treated as a new problem.

another at any desired distance, by means of rigid connecting rods. The boxes are placed in water, which is allowed to heat to about 116° F., then all the wax thoroughly softens. The softened wax is carefully kneaded, into a smooth layer of uniform thickness, covering com-



FIG. 88.—Footprint 1 was taken when the foot was bearing no weight; 2, when the foot was bearing the weight of the body. Note the remarkable spread of the foot in 2; its length and breadth are both increased.

pletely the bottom of the box. The patient is shown how to stand with the feet parallel, and separated by a distance equal to the width at the hips. Then he practices placing one foot, say, his right, squarely upon the ground, meanwhile resting his weight upon the physician; next placing his left upon the ground, parallel with the right; and then, supported by the

physician only enough to prevent swaying, allowing his weight to be borne wholly upon his feet. After two or three trials, this maneuver is easily accomplished. The two boxes are lifted out of the hot water, braced together at the desired distance, and placed in front of the patient. The patient, leaning upon the physician, places first his right foot squarely upon the wax in the right box, next his left foot squarely upon the wax in the left box, and then allows all his weight to rest upon his feet; while doing

so, he is supported by the physician sufficiently to prevent swaying. In about two minutes, the temperature of the wax in the box falls below the melting point, the wax solidifies, and the patient, once more leaning his whole weight upon the physician, then removes his feet from the boxes. In each box, there now remains, deeply impressed in the solid wax, the imprint of the patient's foot as it was when his weight was fully upon it. Into

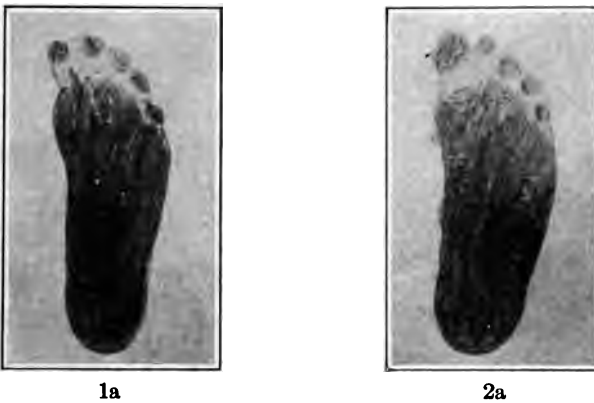


FIG. 89.—Footprint 1a was taken when the foot was bearing no weight. Footprint 2a shows the same foot bearing the weight of the body.

this mold, fluid plaster of Paris is poured and allowed to set. Then by placing the boxes again in water above 110° F., the wax softens, and the plaster casts can be lifted out.

If the temperature of the water be allowed to rise too high, and then to cool too suddenly, the wax becomes hard and brittle. It can be restored to its original plastic state by being again heated, and very slowly allowed to cool. The only precautions necessary in choosing the wax are, first, that the wax be solid at body temperature; and, second, that its melting point be within a few degrees

of the body temperature, so that the patient may not step into wax which is uncomfortably hot.

Plaster casts are now taken of the foot at rest, in the usual way—the leg crossed upon the opposite knee and supported in the position of least constraint, while the cast is being taken.

We are now provided with a plaster cast of the foot in action and a plaster cast of the foot at rest. With these two casts as guides, we build a third. In building this third cast, our effort is to utilize the normal pressure points in the foot and to restore, as far as we can, the normal bony arrangement of the foot, the pressure tripod. For this purpose we use the cast of the foot at rest; add plaster to it at points where we wish to avoid pressure, and scrape away plaster at points where we wish to allow pressure. This modified cast is the model of the foot upon which the brace maker fashions the plate.

The cast of the foot in action shows usually that the faulty position assumed throws the weight on the posterior and outer limb of the tripod. Sometimes, all the weight is borne on the heel; sometimes, on the heel and the internal limb of the tripod; and sometimes on the heel and outer limb of the tripod. In a condition such as the last, the weight falling on the os calcis and the base of the fifth metatarsal and the foot supinated—the following are the main points which guide us in constructing the model for the brace maker. The posterior part of the longitudinal arch, between the tubercle of the os calcis and the base of the fifth metatarsal, is increased sufficiently to throw the foot inwards, and by diminishing the convexity of the heel across the tubercle of the os calcis, the foot is thrown forward, both planes

converging on the inner limb of the tripod, until the weight falls squarely on each. Our aim is to pronate the foot, without pressing on the inner side more than is needed to guide the supinated foot into the proper degree of pronation. The model is made more wide than the cast of rest, but narrower than the cast of action, so as to limit the spread and yet not compress the tissues unduly. The lengthening of the foot is, of course, diminished by the longitudinal arch support. The spread of the transverse arch is also diminished by increasing the concavity between the two anterior limbs of the tripod, and by extending the brace completely across the space.

The brace is a modification of the Whitman brace. The inner lip is curved below the level of the scaphoid; the outer flange is the lower and does not extend so far forward; and the support of the transverse arch extends outwards beneath the four inner toes. When the spread is very great, the inner lip is extended forward to include the first metatarsophalangeal joint, surrounding it completely on the inner side.

No two pairs are exactly alike, each being built on its own model, constructed from the casts of the foot at rest and in action.

The brace is light, accurately applied, and efficient. It supports the foot in action. This support is obtained as the result of study of the foot in action, which has shown us the distribution of the pressure upon the foot. Braced by this plate, the foot is no longer a treacherous, structureless prop, but sustains pressure on the normal pressure points, and affords reliable support from every part of its surface.

This braced foot is encased in a high shoe or boot,

devised by us and made by Mr. Max Deutsch of New York.

A straight last is used. The sole projects three-eighths of an inch around the upper. A hickory wood filler extends from the heel, through the shank, to the front of the sole, rendering the sole rigid and unbending. The heel, wedge-shaped, with the base of the wedge on the

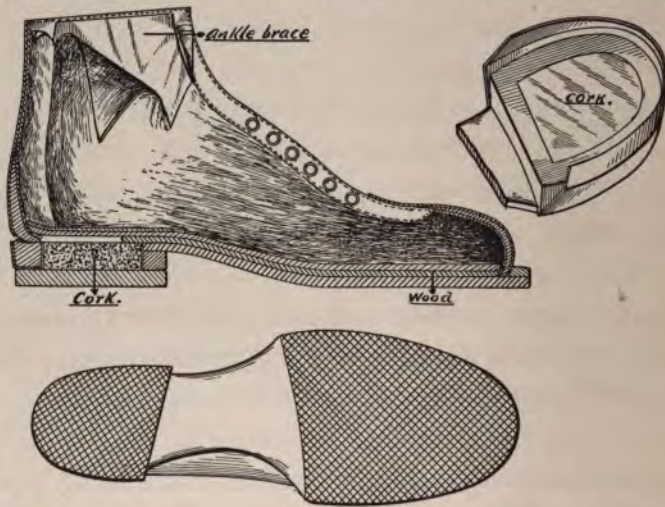


FIG. 90.—The tabetic's boot. Diagrams to show how rigidity, lightness, and support are attained in its construction.

ground, is seven-eighths of an inch high, has its inner and outermost layers of leather, and the two intermediate layers of cork, enveloped in leather. A special feature is made of the snugness of the fit in the longitudinal arch. The box of the shoe is high. The upper is made of calf. Around the ankle, between the lining and the upper, is a small reinforcing leather brace. A thick, soft tongue enables the shoe to be tightly laced. The total weight of a shoe (size 8, on an A last) is just less than fourteen ounces.

The special features of this shoe are, first, its rigidity;

from the moment the foot is put flatly upon the ground, all movement ceases; there is no more oscillation; the foot has an unbending support: second, its lightness which permits the tabetic to walk more with less fatigue; third, the increased ground area of the heel and of the sole, which augments the foothold; and fourth, the ankle support which prevents the ankle from yielding. A patient, thus shod and braced, can use his whole foot,

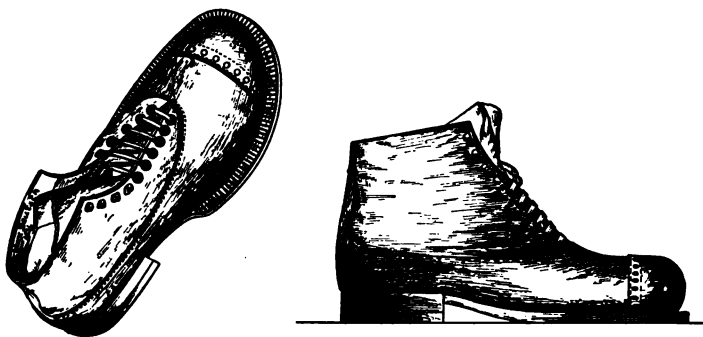


FIG. 91.—The tabetic's boot. Note that the boot rests securely on the ground; its ground surface is quite level and has a larger area than an ordinary shoe.

instead of his heel, as a support. The foot in front of the os calcis, instead of being an unstable, is now a rigid, support. In feet so braced, local and remote pains diminish, and disappear. Shooting pains in the legs seem to decrease both in severity and in frequency. Apparently these pains can be, to a certain extent, reflexly initiated by local irritation in the foot, and the removal of that local irritation diminishes them. Blisters and callous formation disappear. The patient now does not oscillate every time the foot is put down. He walks on a secure, rigid base. He is more stable. He feels more stable. He fears less; and as fear begets ataxia, and ataxia begets fear, his increased reassurance greatly im-

proves his gait. The patient thus supported is soon weaned from the straddle-legged attitude which in

tabetics renders coördinate walking so difficult.

This method of treating the foot is essentially temporary. By means of exercises we increase the tone of the hypotonic muscles until the muscles themselves are capable of maintaining the bones in the correct position. In mechanically supporting the foot we aim solely to maintain the foot in a correct position, and to support it there, until such time as the muscular support may be sufficiently developed to fulfill this function unaided.

In addition to supporting the feet it may be necessary to buttress the knees and the back until the skeleton can acquire its best support, the support of strengthened muscles. Overstretched muscles can neither reinforce joints nor increase in tone. Abnormal mobility of the joints must, therefore, be prevented. But the prevention must not immobilize: movement is essential to muscular health.



FIG. 92.—Showing bandages used as temporary muscles.—“For cases in which abduction and outward rotation of the lower limbs are present, the bandage is tied to the loop on the back of the boot by which the boot is pulled on; then it is carried to the outer side of the leg and wound spirally round the leg. The free end is . . . fixed to a belt . . .” Bandages should not be applied next to the skin.

Nor must the restricting apparatus be heavy or irksome. Rarely a light corset is needed. More often

a simple belt; sometimes, only a bandage. The value of the bandage as a temporary artificial muscle is its adaptability; its drawback, the task of reapplying it daily. The mode of application depends on the case. For cases in which abduction and outward rotation of the lower limbs are present the bandage is tied to the loop on the back of the boot by which the boot is pulled on; then it is carried to the outer side of the leg and wound

spirally round the leg. Two spiral turns, one of which passes behind the knee, usually suffice. The free end is passed once around the waist



FIG. 93.—Showing bandages used as temporary muscles. Bracing of a tabetic whose back is weak and whose knees give. Bandages should not be applied next to the skin.



FIG. 94.—Same patient as in Fig. 85. To show effect of treatment. (Grossman.)

and tied or fixed to itself, to a belt or to a corset. Both legs may need this treatment (Fig. 92).

The shoulders may be braced back by a double spica or a St. Andrew's Cross bandage, from which connecting bands pass to the corset, or belt, or to the bandage fixed round the waist. (Figs. 92 and 93.)



FIG. 95.—Same patient as in Fig. 86. To show effect of treatment. (Grossman.)

By such simple means ataxies reeling on two sticks may be at once induced to walk freely without their sticks in private, and to rely on one stick in public. The mental effect of this bracing is good. The bracing corrects the attitude which is perpetuating the fear, and fatigue of the mental state. It stabilizes the ataxic. It abruptly ends the depression which his progressing incapacity engendered. It enables activity to be carried on with reduced effort. It permits movement in attitudes which have not yet been learnt. It accelerates and facilitates treatment.

Such devices are purely temporary. The braces must be gradually loosened, and, one by one, discarded as the muscular tone improves and as equilibrium and coördinate movement are reacquired. They must be carefully, not indiscriminately, used. Economic conditions sometimes force the tabetic to continue

more active than he should. Mechanical aids are then indispensable in minimizing his enforced efforts, but they must not be permitted to lull the ataxic into neglect of the systematic treatment of his mental state, the only treatment which can control his disability and can render him really effective.

CHAPTER XIV

TREATMENT OF DISORDERS OF MOVEMENT IN TABES (CONTINUED)

Removal of muscular constraint. Easy, confident, and perfect movement founded upon appreciation of postural images. Measures to insure appreciation of postural images. Blindfolding. Training in attention and in capacity for effort. The maintenance of optimal conditions throughout the treatment. Training in attitude recumbent, sitting, "all fours," kneeling, standing. Dosage of postural exercises. Training in movement in these several attitudes as they become stabilized. The cardinal qualities of simple movements. Simple movements. Complex movements. Stabilizing the base for progression. Progressing. Creeping. Kneeling. Walking. Dosage of movement exercises. Walking in public.

AFTER analyzing and measuring the disorder of movement, and after adopting such measures as prevent the further aggravation of that disorder, we proceed to cure it. As disorder of movement arises mainly from deterioration of the mental state below the level necessary for effective action, its treatment should comprise avoidance of the conditions which produced the mental deterioration, rest, training in attention, and the systematic ordering of the mental processes. Mental exercises which afford an externalized result by which the mental process may be judged, controlled and perfected, are essential to the ordering of the mental processes. Improvement in the mental state by these measures is accompanied by diminution of the ataxia. The improvement in movement is a measure of the improvement of the mental state; and that improvement should be such that the tabetic regains the preataxic state.

Movement and attitude are muscular externalizations of mental processes. Accurately to reflect a mental process in muscular action requires a musculature free from all constraint.

There should be no obscuring of the desired, by an existing, muscular expression. The less the admixture of muscular expressions, the more perfectly the movement mirrors the desire. Before evoking a new muscular expression we should remove all that is obliterated of the old. Perfect movement cannot be impressed upon the hidebound tabetic. Before the teaching of movement is attempted, the ataxic, through the rest exercises, must be freed from all the contortions and rigidities which former mental states and exigencies of movement have imprinted upon his musculature.

The blind, directed mainly by their postural, pressure and tactile senses, move coördinately. Normal individuals, in habitual movements, such as walking, are similarly but unconsciously directed (p. 75). When the feeble, delayed and imperfect peripheral after-images of movement mislead the tabetic, he ignores and suppresses them. He criticizes, guides and corrects every movement by aid of vision. During movement his attention is more or less wholly devoted to overseeing his movements. He becomes a visual automaton. When he moves, he thinks only of moving. Unless he carefully scrutinizes his movements, he cannot move.

A watched normal movement is clumsy: a watched tabetic movement is ataxic and involves effort which brings fatigue. The increasing vigilance which movement demands is a constant strain on the tabetic: he may not move without care and foresight; he dreads everything he does not see. Moving by vision is moving with

dread and effort. Such movement is clumsy and destructive of the tabetic's mental capacity. The tabetic must be taught to move easily, confidently, freely and unaided under all circumstances. He must, therefore, be taught to move, not in response to his visual, but to his postural, images. The postural images which he habitually despises and suppresses, he must learn to discern, to appreciate and to utilize. He must attend to his postural images.

To insure attention to these feeble, delayed, imperfect, postural images, external distraction must be obviated by decreasing competition for attention between postural images and images of other sensory fields: a mental state must be procured which is as free as possible from internal distraction, from fear, excitement and other emotional influences detrimental to attention, detrimental to the perception of postural images: and fatigue must be avoided, for the greater the fatigue, the less is the power to attend to the postural images.

In early life, the impulses that underlie sensations of pressure preëempt attention. Pressure soon falls from this eminence as touch and taste associations become more numerous. These, however, grow progressively less important in their turn for they soon are relatively few compared with the myriad of associations from the visual field. As experiences accumulate, the visual associations far outnumber the associations from all the other sensory fields. A visual stimulus, therefore, evokes an image that excites incomparably more associations than a tactile, an auditory, or a postural image. Effortless attention in the normal adult is largely mortgaged to vision.

In the darkness and silence of the night an insignificant

discomfort of the day may become an obsessing pain. Tea-tasters and wine connoisseurs, when they wish to appreciate a flavor, close their eyes as they smell and taste. Everyone has noticed the fixed stare, the rapt gaze, of the unsophisticated being made to marvel.

To obviate external distraction, to enable attention to be focussed upon postural images, irrespective alike of the extent of their demand and of the number of memory images of posture which the tabetic possesses, the tabetic must minimize the competition of his visual images for attention. Absence of visual images from the competition for attention, focusses attention upon postural images and confers upon the blind a perception of posture one-fourth more sensitive than the seeing possess (p. 59).

Ataxic tabetics who have become blind may recover their lost power to perceive posture and become less ataxic. Indeed, we have seen that they may lose spontaneously every trace of incoördination. The feeble, imperfect, and delayed postural images which result from the tabetic lesion contain adequate guidance not merely for the blind tabetic's movements but may suffice even to direct the movement of his ataxic colleagues (Abrahamson's case, p. 99).

To minimize external distraction and to focus attention upon postural images, we teach the ataxic to move blindfolded. To blindfold, we use a mask¹ made of soft washable cloth, which is fixed to the head by means of tapes. Every ataxic should have two masks, for his exclusive use. The blindfolded ataxic is an attentive, a quick, and a successful pupil.

¹Masks and knee guards are obtainable from Kny-Scheerer Company, New York City.

To procure freedom from internal distraction and to avoid fatigue, we use the rest exercises (p. 196). Unless his mental state be tranquil, the tabetic can learn little; unless he has a reserve of strength, his pulse leaps even at the thought of movement; and without the initial training in attention which these exercises confer he is momentarily a prey to fresh distractions.

By blindfolding, distraction is avoided and attention is concentrated. By the rest exercises, a musculature as free as possible from unconscious muscular contractions, and a composed, placid and attentive mental state are obtained. Both mentally and physically, the tranquil, blindfolded ataxic is in the most favorable state for training in normal movement.

Throughout the training, these optimal conditions must be maintained. There must be the most careful graduation of the amount of effort demanded from the ataxic, and when his capacity for effort is reached, rest and recuperation must follow. The effort required from him varies with the difficulty and the duration of the exercises. The easiest must be the initial exercises and their amount must be well within his fatigue limit. To control an ataxic movement is more difficult than to perform the most complex normal movement. So the initial training in cerebral control is gained with respiratory movements, with eye movements, with movements of non-ataxic limbs.

No successful movement is possible unless the relation of the body to its environment is stable; unless there is a fixed point to move upon; unless equilibrium can be maintained during the movement. Movements should be grafted only upon correct well balanced attitudes.

The recumbent, the posture in which the equilibrium

of the body is mechanically and passively assured, is occasionally the only posture which the ataxic can at first maintain; but almost invariably sitting can be achieved.

In the sitting posture he can gain valuable training in stability for the erect attitude. He may at first need an arm chair to support the head and trunk: later, he can sit erect on a stool without back or arms. Next, he practices with one, then with both arms outstretched at the level of the shoulders or held above his head. Finally, in his outstretched hands he supports weights. Similar exercises are later practiced in the kneeling and in the standing postures.

After the sitting posture, "all-fours" is learnt. On all fours, the head should be held well up, the body straight, the thighs at right angles to the trunk, the lower limbs parallel, the feet slightly dorsiflexed, and the toes on the ground. The feet at first may need the bracing effect of a friendly wall and the posture may require aid for its maintenance. Such aid should be unsparingly given and only withdrawn as improvement warrants. In the all-fours attitude, the amount of aid required is usually very slight and is soon dispensed with.

After stability on all-fours is gained, kneeling is practiced. Besides swaying, the tendency here is to bend forward from the hips. Great care should be used to procure a proper upright attitude, with the head and trunk erect. After acquiring first sitting, next all-fours, and then kneeling, the tabetic finally learns to stand. Stability in the erect posture depends largely upon the thoroughness of the preliminary training of the head and trunk and lower limbs in the sitting, all-fours, and kneel-

ing postures, and upon the adequacy of the mechanical bracing of the foot and lower limbs.

The length of time for which a posture is maintained should be carefully measured. At first the tabetic may be able to preserve his posture only for a few seconds. Succor should always be immediate and effective so that no cause for unnecessary fear shall complicate the task. When the patient is placed in a posture a warning "Get ready," followed after an agreed interval by "Now," marks the withdrawal of aid. Then counting is commenced, the patient having been told to maintain his posture for a definite time. With repetition this time is gradually lengthened.

The dosage of the postural exercises, their difficulty, frequency and duration, should be scrupulously regulated; should always remain well within the fatigue limit; and should only be increased with the raising of that limit. Every posture should be learnt blindfolded and should be practiced till it becomes effortless.

Contemporaneously with the postural exercises, movement should be taught. As no movement is permissible except in a correct, well balanced posture, and as the recumbent may be the only perfect posture, the movement exercises must often be first practiced recumbent. As more difficult attitudes are stabilized, movement is commenced in them. Thus, while the erect sitting attitude is being learnt, movements in the recumbent posture are practiced; while the all-fours attitude is being learnt, movements in the sitting attitude are practiced; while the erect standing position is being learnt, movements in the other, easier, and already acquired attitudes are practiced.

The simplest movements are the least disordered and

they are the easiest to perfect. They are, therefore, taught first. The perfection of a simple movement depends upon the perfection of its cardinal qualities, strength, direction, extent and rhythm.

For training in strength, resistance exercises are employed. These resistance exercises begin before movement is taught. The patient's head or trunk, thigh, knee or foot is placed in an attitude which he is exhorted to maintain for a given period, against the physician's efforts to move it. The period should at first be short, two or three seconds, and should be gradually increased to ten seconds. The timing must be audible. These exercises are varied by the patient's attempting to move against the physician's immobilizing resistance. The resistance exercises are a great aid to postural sense appreciation.

Most of the weakness of tabes is mental, not muscular. Even when it is muscular, graduated, passive and active resisted movement is its best cure. But massage and electricity may usefully aid. Massage at first should be light and should be applied only to a part of the body, for say, five to ten minutes; later, it should progressively increase in strength, and should gradually extend to comprise the whole body. Electricity may aid the tone of the muscles. Its physical consequences may be insignificant, although its mental effect is conspicuous. It should be mild and painless. With galvanism, large electrodes must be used to avoid burning. Mild electrical treatment lends a useful informality to psychotherapeutic enquiry. I seldom use either massage or electricity.

The beginning and the end of every movement should be definitely fixed. The series of postural images which comprise the movement should be invariable. The more

often the same images are linked the readier and the more inevitably do they associate with one another and not with postural images unrelated to the series, and the more rapid and perfect becomes the movement. Frequent repetition engraves these images and their sequence upon the tabetic's memory.

Instruction in the direction and extent of a movement begins by the physician's passively performing the movement upon the blindfolded tabetic. Then the tabetic resists the passive movement. Next the tabetic's attempts to perform the movement are guided. The guidance is gradually diminished as the movements gain in accuracy. The difficulty of a simple movement varies with its range. The initial movements should be of small extent.

For training in rhythm and rate, vocal timing is excellent. A loud ticking watch or clock is useful. But best is the ordinary metronome, obtainable at all music dealers. Every ataxic should have one for his private use. All movements should at first be performed slowly, but not too slowly. The initial optimal rate can readily be ascertained by trial. This rate must gradually quicken, but not at the expense of efficient action.

A movement is not perfect till it can be well performed at all speeds, against resistance. After an interval which varies with the individual, and which is longer the more complex the movement, the movement is so acquired that the eliciting of the desired, in association with the existing, postural images, excites in order the images which link them, and the movement is performed with a minimum of effort.

In our appreciation of the body's posture, the dominant images from the head and trunk are more important

than those from the limbs; those from the legs, than those from the arms; and those from large, than those from small joints. Most rapid progress is attained when these various sections are trained in the order of their importance.

To teach the recumbent patient to move, we first relax him. After general relaxation has been practiced, attention is specially focussed on the head.

The recumbent ataxic with the muscles of his head and neck relaxed, the shoulders supported on a sandbag and the head extended, is instructed to count slowly while the physician at a uniform rate repeatedly moves the head to a position of flexion, pauses, and then returns it to the original extended position. Next, the patient lightly but steadily resists the passive movement. Then, the movement is practiced with the patient performing and the physician aiding and guiding. The physician's aid gradually lessens as improvement occurs, until the movement is executed by the patient alone. Finally the movement is made against resistance. A movement which cannot be made against resistance is still faulty.

Every movement is timed first by the physician, then by the patient, finally by the metronome. Movements at first are slow: as they are perfected they are quickened; finally, practice is obtained in varying the rate with that of the metronome. This training in time is essential to smooth, easy movement.

The trunk is similarly exercised. In the recumbent posture, while exercising the trunk, at first, the tabetic has his feet fixed, a rope in his hand, and the teacher's aid. Gradually these auxiliaries are eliminated.

The training of head and of trunk may be varied by

changing from the recumbent to the sitting and from the sitting to the recumbent posture.

Besides head and trunk, limb movements may be practiced in the recumbent attitude.

After general relaxation, attention is directed especially to relaxing one limb, say the right lower. Relaxation of the hip, knee and ankle are then in turn procured. The hip is first dealt with, next the knee, then the hip and knee, then the ankle, and finally hip and knee and ankle together, so that the degree of muscular relaxation in the chosen limb shall be as great as is obtainable.

The postural sense loss in the hip is less than that in the knee, in the knee than in the ankle, in the ankle than in the toes. The greater the number of surviving postural impressions in any joint, the greater is the ease with which movement at that joint can be made coördinate. Training in movement should start at the hip, then proceed to the knee and finally to the ankle.

At first, all teaching should be confined to flexion and extension. The legs may be supported at right angles to the thighs, on a chair or board, while active unaided flexion and extension of the thigh on the abdomen is practiced. A light posterior splint may usefully immobilize the knee when first flexion and extension of the lower limb are taught. Lateral and rotatory movements may need to be controlled by bandages. These mechanical aids are soon discarded.

After the hip, the knee movements are practiced. There the patient's task is simple, for only flexion and extension are possible. Also, as less strength is needed, the tabetic does not tire so quickly from knee as from hip movements. The knee movements are practiced by allowing the leg to hang over the end of the couch; the

recumbent tabetic exercises first on his back and later face downwards.

After the hip, the knee, and then the ankle, first of the same limb then of its fellow, are educated in flexion and extension. In every exercise, first relaxation, next passive, next again passive resisted, then active guided, later active unaided, and finally active resisted movements are practiced. When the hip movements are acquired, then the movement exercises begin with hip movements: when knee movements are perfected, the lesson begins with hip, then proceeds to knee and last to the yet unlearned ankle movements. The repetition of a movement which can be well performed is a good introduction to the learning of the yet imperfect movement.

The movements acquired in the recumbent are practiced in the sitting, in the all-fours, in the kneeling, and in the standing attitude, as these attitudes are successively stabilized.

Complex movements, such as creeping and walking, are compounded of simple movements. When the direction and extent of the component simple movements have been acquired, these movements should be practiced in sequence so that the series of postural images which belong to each of the several components may be linked together. Then the eliciting of any one series elicits the images of the allied series in order. The tabetic acquires functional facilitation among his postural images.

In the recumbent and in the sitting posture, the movements of the limbs necessary for creeping and walking should be practiced. If such movements cannot be performed when the patient is spared the effort to balance,

much less can they be performed when, in addition to the movements, the patient must also balance.

Progression demands the power to maintain posture not merely in the usual stationary attitudes but also on the diminished base of support which is inherent to progression. Stability upon this diminished base is a necessary preliminary to progression. Progression is acquired first in the most stable posture for progressing, the creeping posture, a posture which involves active movements only at the hip and shoulder; then in the kneeling; and finally in the standing. The first steps should be small and slow. To insure direction in blind-fold progression, I use strips of carpet or of linoleum. For novices, strips three to four feet wide are necessary. The width decreases as the movements improve.

The knees in creeping should be protected. I use knee-guards somewhat similar to those used by carpenters. These knee-guards consist of two rolls about nine inches long and one inch in transverse section, made of stout cloth stuffed with hair, which are fixed parallel, at a distance of about one and a half inches, upon a layer of thick carpet. Above this carpet layer, between the parallel rolls, the patient's knee rests, and thus is prevented from touching the ground. This knee-guard gives good protection. It also gives considerable support for it broadens the kneeling surface and prevents the patient from rolling. A soft continuation extends from the guard about two inches upwards. To this continuation and to the guard, tapes are sewn. Every pair of tapes has a buckle by which it is fixed around the limb. The tapes keep the guard in place (Fig. 96).

Shoes must be worn while creeping, in order to protect the toes.

In creeping, a step involves the temporary loss of one of the four supports; hence, creeping is possible only if the patient can balance for the duration of that step on the remaining tripod. The patient first learns to throw his weight in turn on the four possible tripods, the left hand, left knee, right knee; right hand, left knee and right knee; left hand, right hand, left knee; left hand, right hand, right knee. Lifting first a hand, and later a knee, from the floor is then practiced till stability can be maintained on any of the four tripods. When stability on all-fours is gained, and stability on three points, then one hand is moved forward, the weight redistributed, and then the hand is moved back. The soles of the feet should rest against a wall; from the wall, the foot moves forward when the thigh is flexed and the knee is lifted off the ground; to the wall the foot is returned, on completing the movement. After exercising one side, the other side is exercised. Later the maneuver may be reversed; first the hand is moved backwards, and then the knee.

Creeping is first performed, moving limb by limb at command. Thus, at the word "One," the right hand is moved forward; at "Two," the right knee; at "Three," the left hand; at "Four," the left knee. The usual tendency is for a small step to be made by the hand and a large one by the knee; both hand and knee must move forward equal distances. The knees in move-



FIG. 96.—"The knees in creeping must be protected." Any efficient knee guards may be used. Those shown here are satisfactory.

ment tend to be abducted and circumducted; they must be moved straight forward. The feet fly from the ground, and also separate widely. They must be kept parallel and the toes must remain on the ground. It is sometimes easier to begin with backward creeping.

When proficiency in creeping is gained, then kneeling is practiced. Next, the weight is balanced first on one knee, then on the other. Then one knee after the other is momentarily raised; and then small forward steps are made. At first the tabetic merely shuffles the knees slowly along the ground; later he moves more freely and quickly; but always he remains erect.

Finally walking is taught. The blindfolded patient, standing facing and near to a wall, for reassurance and possible support, throws his weight from one foot to the other by command for a measured length of time. This he practices till he can change his balance from foot to foot as fast as the metronome can beat. Next he practices rising on the toes of one foot, then of the other and then on the toes of both feet simultaneously. Now, not changing his place, he lifts one foot after the other from the ground, at first cautiously, slowly, but soon with increasing speed. Next, one foot is advanced and the weight is thrown forwards and backwards from the rear foot to the advanced. Then, the rear foot is brought alongside the advanced. So, advancing with, say, the left and slowly shuffling the right alongside, progression starts. Then the right is made the advancing foot. Next he advances either foot by command. Then he advances the moving foot from slightly behind the stationary to slightly in front of it; and back again; changes the moving foot, and takes successive steps: soon he increases the number of steps; lengthens the stride; walks back-

wards; sideways; and turns. Later, he walks up and down increasingly steep inclines; negotiates steps, at first shallow, then deeper, then unequal; walks heel and toe; carries weights in the outstretched hands; runs; catches a small medicine ball—there are few limits to the ataxic's capacity to learn.

These movement exercises are merely a means to procure orderly mental action, a means to train attention, a means to inculcate cerebral control. They become not only useless but actively injurious when they are practiced to the extent of inducing severe fatigue. Every movement requires effort. To avoid fatigue the exercises must involve the minimum of effort. This minimum is achieved by devising exercises and safeguards so as to eliminate fear from practice, and by carefully grading the difficulty and the duration of the exercises. The only fatigue permissible to the ataxic tabetic should be that which comes from moving repeatedly and with care.

Certain bodily changes accompany effort. That most commonly used as an index to the amount of effort is the pulse rate. To exercise till the pulse rate reaches 110 per minute (or, as I recently saw advocated, 140), is to retard progress and to incur risk. Above a certain pulse rate, usually above 100 per minute, effort is not profitable. That rate must be ascertained by observation of the individual tabetic. When it is exceeded, the movements deteriorate and further exercising wastes time and causes futile strain. The need then is to practice not movement but rest. Five minutes spent on rest exercises will refresh the tabetic, relieve his mental tension and reduce the pulse rate to normal. Then, the practice of movement may usefully recommence.

For every patient, the most profitable ratio of move-

ment to rest exercises should be determined by experiment; and the dosage of the exercises must conform to this ratio. This ratio is not constant; it varies from day to day and is greater at the beginning than at the end of a lesson in movement. When during the course of the lesson movement does not improve, general relaxation should be given and then a fresh start made.

With judicious alternation of movement and of rest exercises, I have worked profitably for two hours at a stretch with a patient. Every session should end with rest exercises which return the tabetic to the physical state in which the session began. When owing to pain or crises, or other untoward circumstances, the tabetic is depressed or fatigued, there should be more rest than movement in the exercises. Not the patient's complaints, but the patient's reactions to the exercise should determine the program. Pain usually diminishes and disappears if the practice is tactfully conducted. The treatment of the disordered mental state which induces ataxia is also the treatment of the disordered mental state which magnifies pain.

When the tabetic is mentally competent to move coördinately in spite of all ordinary emergencies in sheltered life, his activities should be broadened. He should be taught to walk freely, confidently, and unaided in public, at street crossings, and at change of level and of surface. These tasks should, like all other tasks of the tabetic, be graded in difficulty and in duration.

The extent to which the ataxia will improve by this treatment is always considerable and often remarkable. In slight cases a complete return to the preataxic stage is certain. In recent cases, cases which have been ataxic for a year or less, similar success is likewise easily at-

tained. No matter how long standing the case or how severe the ataxia, improvement is inevitable if the patient be free from organic mental disease.

I believe there is no sane ataxic who cannot be taught to move coördinately. When measurement shows that improvement halts, either the treatment is being unduly hastened, or extraneous circumstances are preventing progress. Examination will at once disclose if the treatment is at fault; if a fundamental posture or movement has been so imperfectly acquired that all subsequent movements of which it forms a part must share its imperfection. If the treatment be not at fault, then the tabetic's economic or domestic life is. Economic conditions are a factor only in the ambulant ataxic, the ataxic who must earn his living while being treated. The patient's improvement will depend upon the extent to which we can mold his circumstances to his benefit.

Blindness, arthropathies, fractures, and muscular atrophy are not contraindications but incentives to treatment. One formerly helpless ataxic who demonstrated the cure of his ataxia to the students of the International Extension Course, subsequently developed a Charcot ankle and knee, and fell and fractured his tibia; but, with some mechanical aid, he persevered in his efforts and manages still to walk well enough to earn his living. The possessor of the fractured tibia, shown in Fig. 60, formerly ataxic, now walks excellently and pursues his calling uninterruptedly (Fig. 95).

The length of time necessary to restore an ataxic to the preataxic stage varies with the individual and with the degree of ataxia. The more intelligent and industrious he is, the quicker he learns. The less ataxic he is, the less is his difficulty. Five lessons may be adequate

to teach the rest exercises. If Rombergism alone is present little more teaching is necessary to procure equilibrium.

With cases of average severity under constant supervision, one month's training is usually adequate. If the ataxia developed suddenly it is usually quickly removed. Ambulant cases, and cases long neglected, cases which have gradually advanced for years, take longer. But so long as periodic measurement shows steady improvement, the encouraged tabetic cheerfully strives on, and progresses as rapidly as his capacity and his circumstances permit.

Permanence, not speed, should distinguish the cure. When, by the treatment of the mental state, the tabetic is made once more preataxic, he must be maintained preataxic. His daily efforts must be reduced to his capacity. He must avoid occasions of undue mental strain. He must remain for a time subject to the vigilant medical guardianship which every preataxic tabetic requires. But having once conquered ataxia, the tabetic is learned in the causes of his disability; he can never again sink to the depths from which he has emerged. Disturbances in his gait now induce in him not fear but study. He reports his transitory lapse together with its cause. He should then revert to the practice of the very simplest movements. A day or two usually suffice to stabilize him again. When I returned from the war, three of my preataxic tabetics, who had long ceased to need my care, reported that soon after they heard of my departure their movements became somewhat disorganized. This disturbance endured in one case for nearly a week, in the others for several days. All three completely recovered by their own efforts.

With the disappearance of the ataxia, pain and crises and other symptoms diminish or disappear. The relief of ataxia is merely incidental to improving the mental state, to equipping the tabetic mentally, so that he will be able to live with his irreparable structural changes in the minimum of discomfort.

CHAPTER XV

REVIEW OF TABES

Afferent nervous system invaded in disease styled *tabes dorsalis*, or locomotor ataxia. Disturbance of vegetative, non-vegetative and sensory function. "Locomotor ataxia" misleading; "*tabes dorsalis*" useless; "syphilitic neuritis of posterior roots" suggested. Diagnosis and course of invasion. Division into preataxic, ataxic, and paralytic stages a psychological not an anatomical division. Drug treatment of *tabes*. Reciprocal relations of symptoms and mental state in *tabes*, and interdependence of symptoms. Treatment of the symptoms through the mental state. Its ease and effectiveness. Treatment of the mental state through the symptoms. Former methods, Frenkel, Goldscheider, Förster. Limitations of Frenkel's method. Development of present method. Value of present method in cure and in prevention. Conclusion.

THE first symptoms of invasion of the central nervous system by the *spirochaeta pallida*, the spirochete of syphilis, usually arise from implication of the vegetative nervous system in the pupil. The invasion then spreads to involve the vegetative nervous system elsewhere, to produce lightning pains, or bladder, or stomach, or other visceral trouble. At the same time it may affect common sensory fibers in the posterior root, or special sensory nerves such as the optic.

The lesions of the afferent sensory mechanism in *tabes* cause fewer, feebler, and delayed sensory impulses. These sensory impulses are of two kinds, non-sentient and sentient. The non-sentient underlie vegetative and non-vegetative reflex function: the sentient, perception.

Local disturbances of vegetative reflex function may be inconsequential for there is great local autonomy in

the vegetative nervous system. But the cumulative effect of such disturbances is to disorganize the balance of the system as a whole, to disorganize general vegetative functions, such as metabolism and defense, and to lead to vagal cataclysms, such as gastric crises.

The amount of disturbance of the non-vegetative or motor reflex function in tabes varies with the site, extent, rapidity of development, and age of the lesions. Its amount is often unimportant, for there resides in the reflex mechanism a vast power of integration. One gets an occasional opportunity to realize the extent of this power in viewing the profound spinal changes which in life produce insignificant manifestations in cases of, for example, disseminated sclerosis.

Upon the attention devoted to the surviving sensory impulses, as well as upon the extent and severity of the lesions, depends the amount of loss of sensory perception. The simpler the sensation, the more accurately does the failure of perception reflect the tabetic destruction.

So long as the dominant symptoms arise from lesions of the afferent nervous system, whether vegetative, or spinal, or cranial, the disease is now styled locomotor ataxia or tabes dorsalis. As the lesions affect not the motor but the sensory system and as ataxia need not and should not complicate it, the title locomotor ataxia is misleading. Excepting habit and use, there is as little justification for the continued survival of the Hippocratic term tabes dorsalis as for the persistence of words such as metasyphilitic and parasyphilitic. The employment of meaningless, archaic names for syphilitic infections of the central nervous system leads to regrettable confusion in thinking, in writing, and in teaching. Revision

and standardizing of the nomenclature of syphilitic affections of the central nervous system urgently await action by an influential congress of neurologists.

The diagnosis of tabes depends upon the discovery of the presence of syphilis and of signs of implication of the afferent nervous system. The minimum amount of implication usually considered tabetic is implication of vegetative and of common sensory fibers, an implication which in the posterior nerve root occurs as the result of the invasion of a single site. In absence of any *ex cathedra* pronouncement to the contrary, a disease in which a primary syphilitic lesion of the posterior nerve roots causes the dominant symptoms may be considered as tabes.

The spirochete attacks no site exclusively, but invasion early becoming dominant in the posterior roots, or in the optic nerve, or in the cerebral cortex, respectively, tends to follow a certain course. The designation of the dominant site somewhat presages the future of the invasion. At any site, pathological processes are named according to the tissue which is essentially attacked. Thus, pneumonia remains pneumonia although pleurisy supervenes. The essential lesion in tabes seems to be a syphilitic neuritis. Should it not be so styled? Until we know whether the pathological change is primarily interstitial, as Nageotte contends, or primarily parenchymatous, as Stargardt seems to show, the term "primary syphilitic neuritis of the posterior root" would have at least the merit of describing the cause, the dominant site, and the nature of most of the syphilitic invasions of the central nervous system which we now call tabes. So long as the posterior root lesion produces the dominant symptoms, the disease still merits

the description. If optic neuritis, or encephalitis, or poliomyelitis, assumes the dominance it becomes entitled to designate the invasion.

Beyond the facts that syphilis beginning in the posterior roots tends to spread first among them, and that the longer the invasion lasts the more general is the spread, we cannot foretell its course. The old division of the disease into preataxic, ataxic, and "paralytic" stages is founded not upon a pathological but upon a psychological basis. The ataxic tabetic is merely one whose mental treatment has been neglected, the "paralytic" one who has ceased to strive against his infirmity and has surrendered activity rather than incur the exertion which activity demands.

Before neurologists could diagnose tabes, except in its most aggravated forms, Erb wrote "*Ueber alle ist der Stab gebrochen.*" Brissaud later remarked that the disease was growing milder; and Raymond, that pains and ataxia were becoming less severe. Now we recognize that about one-seventh of all cases are abortive: and that tabes is a disease which is practically without lethal power.

As tabes begins about the age of thirty-seven and lasts on an average until more than fifty-three years, the tabetic has a life expectancy equal to at least two-thirds that of the average healthy man. Yet the force of tradition and the pessimism of ignorance still combine to make of tabes an affection as fatal as cancer, as dreadful as leprosy. I know of two tabetics who committed suicide; they were both medical men, were both victims to false teaching.

As life is neither menaced nor much shortened by tabes, the tabetic is an ideal patient to treat.

Successful treatment of syphilis in tissues such as those of the central nervous system, where lesions are irreparable, lies in preventing the invasion of these tissues by aiding the body to kill the spirochete ere it attacks them. Fournier with all his skill in antisyphilitic treatment, would predict from the banal lesions the later occurrence of tabes, and Ehrlich expressly stated that salvarsan was merely an adjuvant to the human defense substances. And Warthin has shown post mortem the presence of spirochetes in the central nervous system of intensively treated cases of nerve syphilis.

We cannot by metallic substances, alone, surely kill the invading spirochete. Modern therapy pays too much attention to the spirochete and too little to its host. Our present efforts seem not so much to reinforce as to attempt to replace human defense by antisyphilitic remedies. These efforts have not succeeded. Our tasks now are to learn to develop and strengthen human defense and to recognize and combat imperfect defense against the spirochete before its evil and irreparable consequences arise.

The correlation of biochemical action upon which defense depends is a function of the vegetative nervous system. I have suggested as a cause of imperfect defense, inadequate stimulation of the vegetative nervous system owing to the mildness or absence of the pretabetic lesions. Perhaps our haste to banish ordinary syphilitic lesions by drugs may contribute to increase the frequency of nervous syphilis.

The importance of mental stress in conducing to tabes lies in the disturbance by mental stress of the control of the vegetative system over the chemical confederacy. The progressive nature of spinal tabes and the abortive

tendency of optic tabes are partly due to the implication of the vegetative nervous system in the first and to its immunity in the second. Neither the transient inhibition of the growth of the spirochete nor the indiscriminate general excitation of the vegetative nervous system, exerted by the metallic substances which we now employ as antisyphilitic remedies, but the development and reinforcement of the natural human defense against the spirochete by well directed stimulation of the vegetative nervous system, with specific, unorganized, chemical substances, such as vaccines, will ultimately afford the cure for which we seek. This cure is now unknown to us, mainly, I think, because its quest has been neglected in favor of the pursuit of the philosopher's stone of medicine, the drug which will totally destroy the spirochete in every human tissue.

In default of any agent of proven power to cure or to arrest the syphilitic invasion, we are forced to treat, not the structural results of that invasion, but the tabetic's reaction to them. For this purpose we have examined the various symptoms to determine the relative importance of the structural and psychic elements in their production; and we have studied the mental state, together with the influences favorable and unfavorable to mental action. We found that the mental state and the tabetic symptom are reciprocally related and that through the mental state the symptoms are interdependent. As a necessary corollary to these relations, improvement of the mental state relieves the tabetic symptoms and relief of any tabetic symptom improves the mental state and alleviates the other symptoms.

The principles on which I have founded the treatment of the mental state are, I think, basic. The method I

have described of applying these principles is effective. But many other measures of training in cerebral control may be devised. Any mental task neither too complex nor too novel, whose performance can be checked and measured, which can be repeated both unchanged and with regulated complexity, will prove serviceable. Nor need the instruments I have mentioned be used. These are of value where the similarly sick congregate, for they simplify work and permit greater accuracy in treatment. But a hand mirror may take the place of the cephalograph and of the modified perimeter. Lines chalked on the wall or marked by tapes may guide the eyes in the direction and extent of the movement. Striking out certain vowels or consonants with a pencil, from the columns of a daily newspaper, will afford a useful substitute for reckoning tests. No appliances are needed. The one requisite is the interest of the physician.

Properly directed treatment brings immediate results. The effect of every hour's instruction is evident in improvement. Skill is less necessary than systematization in the treatment. From the outline I have given every physician can effectively treat a tabetic. Every physician can become a center round which the incipient tabetics may gather for that guidance and training which will alleviate and may arrest the disease. No extensive experience is necessary. An intelligent study of a single case will fortify the physician against most of the problems he may subsequently encounter. And when a difficulty arises, consideration of it in association with the essential principles which govern the treatment will provide a solution.

As motor disturbance, especially ataxia, is to most tabetics the chief burden of the disease, and as the prob-

lem of voluntary movement particularly interested me, I used the treatment of ataxia to exemplify in detail the relief and cure of a tabetic disability.

Disturbance of voluntary movement is neither an immediate nor an inevitable nor a direct effect of the tabetic lesions. It is an effect in which the tabetic lesions play a part but in which deterioration of the mental state plays the major rôle. So long as the mental processes of the tabetic reflect with sufficient intensity his desire to move, the structural changes remain masked. When the mental state deteriorates to a level at which orderly movement is no longer possible, the resulting ataxic movement is compounded of the desire, the mental state, and the reflex defect.

Ataxia, which is in large measure merely a result of deterioration of the mental state, unfortunately, has hitherto been the first sign to induce the physician to treat the mental state. The nature of the symptom providentially directed the mode of treatment. But the physician tried to bring order out of ataxic movements without realizing that his real purpose was to bring order to chaotic mental processes. Hence, to his own hindrance and to the patient's detriment, the physician was ignorant of the help which lay to his hand.

The apostle of the ataxic tabetic is Frenkel. To his genius we owe, if not the discovery, at least the practical application of motor therapy.

Since 1897, he and his disciples—greatest among whom is Förster—with untiring persistence and admirable skill have striven to educate medical opinion on behalf of the tabetic. To Frenkel and his co-workers, medicine owes the present universal recognition that the ataxic can be substantially aided. Goldscheider, 1899, in a

book of a few score of pages, a masterpiece of clear thinking and of orderly exposition, systematized Frenkel's method and first clearly taught that upon the perfection of the primary qualities of direction, extent and rhythm depends the perfection of the simple movement which is the source of the perfection of all complex movements. To Frenkel, Goldscheider, and Förster is due practically all the effective aid hitherto given to the tabetic.

In common with most who practiced the Frenkel method, I was often disappointed with its results. Although by its aid many ataxics could laboriously be brought to a fair degree of motor efficiency, many could not. The latter might acquire some dexterity in moving in the recumbent posture, and they might slightly improve in walking, but they then improved no further. Such were the timorous and those who by the results of their efforts were not encouraged to persevere. Failing vision and arthropathy were stated by Frenkel to contraindicate his treatment. I never succeeded by the Frenkel method in educating an average ataxic to walk without aid. Nor does Frenkel state that such success is probable. The freedom of the blind tabetic from ataxia and the complete recovery from ataxia which may spontaneously follow the establishment of blindness, led me to teach ataxics to move blindfolded in the hope that the tabetic would thereby attain greater perfection in movement. One of my early patients was an ataxic who had derived great benefit from Professor Frenkel's personal care, but who being seated one night in a restaurant when the lights suddenly failed, soon found himself sprawling helplessly on the floor. Blindfolded teaching promised, therefore, more perfect and more useful move-

ment. This promise has not only been fulfilled, but blindfolded moving has, moreover, proved easier to learn than moving by vision.

My former colleague, Professor Wachsmann, without knowledge of my work, also conceived the idea of teaching ataxics to move blindfolded. I demonstrated to the International Extension Course in 1913, together with two of my own cases, a tabetic whom he had thus taught to move excellently.

To combat the depression which prolonged practice in the recumbent posture caused, to obtain an easy transition from movements in stationary attitudes to progression, and to strengthen and correlate the head and trunk movements, I introduced creeping and kneeling exercises. To insure easy movement, I next used passive exercises to remove muscular constraint. To calm and to rest the excited, fatigued tabetic, after movement, I found that these passive exercises together with breathing exercises were effective.

The success of the rest exercises, not merely in tabes but also in cases of muscular, vascular, and mental tension (Sorapure and Maloney), in spasmodic muscular affections of functional origin (Abrahamson and Pollen), and in chorea (Grossman), and observations of the effect of fatigue and fear upon ataxia, emphasized the mental factor in ataxia to a degree which I had hitherto not realized. Then there quickly followed the systematizing of the treatment: its application not only to the relief of ataxia, but also to the relief of other tabetic symptoms; next its use to prevent these symptoms; and finally its employment in the treatment of syphilitic neurasthenia.

Those with fracture, with arthropathy, with muscu-

lar atrophy and with failing vision are in as urgent need of treatment as their more fortunate fellows and their special disabilities are rather incentives than contraindications to treatment.



FIG. 97.—This patient suffers from the ununited fracture of the tibia shown in Fig. 60, but in spite of that defect he was successfully cured of ataxia. (Grossman.)

As a cure of ataxia the treatment I have outlined has proved speedy and efficacious, not merely in my experience but in that of others. Dr. Sorapure, Dr. Grossman and Dr. Wolf have demonstrated to various medical societies former ataxics whom they have by this method taught to walk confidently and well. Drs. Grossman and Wolf have published some of their cases. Dr. Grossman and I have further publicly demonstrated cases first before and again after treatment had removed the ataxia. The amount of improvement which must occur before an ataxic, blindfolded and unaided, can successfully undergo the ordeal of a public demonstration will be appreciated by all who have any ex-

perience of the ataxic tabetic.

Of the value of this treatment in preventing ataxia I have much less experience. But no case under the care of any of the physicians who have used this method or under my care has developed ataxia. Economic conditions may force the tabetic to continue to toil beyond his

strength; business reverses may sway the tabetic more powerfully than therapy can counterbalance; family dissensions, accidents, chills, or other disadvantageous happenings may impose a limit on the success of the therapy; and ataxia may develop. Regret in such circumstances will be softened by time, the time by which the preataxic stage has exceeded its average duration; by the certainty that the disaster can be speedily retrieved; and by the knowledge that its memory will ever after exert a salutary restraining influence upon the tabetic and upon his associates.

The treatment of the ataxic tabetic differs in no essential detail from the treatment of the preataxic tabetic: and the treatment of the preataxic tabetic is identical with that of the syphilitic neurasthenic. If physicians will only realize the unity of purpose which governs the treatment of these conditions, much of the burden may be lifted from the shoulders of the victims of syphilis of the central nervous system.

The results are more gratifying than any I know in the realm of therapy outside of surgery. To raise the hopeless bedridden tabetic; to make him walk again; to place him once more not only in relation to but in competition with his normal fellows; to arrest the wretched tabetic's progress in ataxia; to lead him back to confidence, to stability, to ordered movement; and, greatest of all, to prevent ataxia, to safeguard the tabetic from the misery with which his ignorance soon endows his disease—these services are not only a duty which the medical man owes to his patient, they are services which humanity claims as a right.

The victims of tabes are the spirochete's prey among

the intellectual. No country can afford to allow their valuable lives to be frittered away for lack of a few weeks of systematic treatment. In any community their number is seldom sufficient to arouse public interest to the point necessary for action on their behalf. The founding of a small national hospital for their treatment and for their further study, would be, indeed, beneficent.

BIBLIOGRAPHY

- ABADIE, J. Constitution d'un schéma sémiologique du tabes, *Rev. neurol.*, 1911, p. 787.
- ABRAHAMSON AND POLLEN. On the Treatment of Spasmodic Muscular Affections of Functional Origin by Maloney's Method. Paper read before the New York Neurological Society, April, 1914.
- APELT. Die Bedeutung cytologischer Untersuchungen der Cerebrospinalflüssigkeit für die Neurologie, *Monatschr. f. Psychiat. u. Neurol.*, No. 20. *See also* Nonne's Syphilis und Nervensystem.
- BAILEY, PEARCE. The Effect of Early Optic Atrophy upon the Course of Locomotor Ataxia, *Med. Rec.*, Nov. 14, 1896.
- BARKER, LEWELLYS F. Monographic Medicine, New York, D. Appleton & Co., 1916, vol. iv.
- BEAUSSART. Tabetiques et paralytiques généraux conjugaux et familiaux, *J. de Neurol*, Brussels, 1910, p. 341.
- BELIÈRES. Quoted by Beaussart, *loc cit.*
- BENEDICT. Quoted by Sachs, Strauss and Kaliski, Modern Methods of Treatment of Syphilis of the Nervous System, *Am. J. Med. Sci.*, Nov., 1914.
- BENEDIKT, MORIZ. Ueber Aetiologie, Prognose und Therapie der Tabes, *Wien. med. Presse*, 1881, p. 101.
- BENENATI. A proposito del rapporto fra cecita ed atassia in due casi di tabe, *Riv. crit. di clin. med.*, 1901, p. 73.
- BERGER. Quoted by Inglerans, *loc. cit.*
- BIEDL, A. Innere Sekretion, 2nd ed., Vienna, 1913.
- BOUCHAUD. *Gaz. hebdomadaire*, Nov. 19, 1892.
- BRAMWELL, BYROM. Analysis of 155 Cases of Tabes, Brain, 1902; *Proc. Neuro. Soc.*, June 27, 1903.
- BRISSAUD. Leçons sur les maladies nerveuses, Paris, 1895.
- BRODSKY. L'influence des événements revolutionnaires sur la cours du tabes dorsalis, *Cong. d. med. russes*, April 23, 1907.

- BROWN, GRAHAM. Ataxia, a Symptom. Edin. Med. J., July, 1912, p. 15.
- BROWN-SÉQUARD. Lectures on Paralysis of the Lower Extremities, Phila., 1861.
- BURR, CHAS. W. The Cause of Death in Tabes, J. Nerv. & Ment. Dis., New York, March, 1912, vol. 39.
- BUZZARD. Optic Atrophy in Nervous Diseases, Brit. Med. J., Oct. 7, 1893.
- CAMP, C. D. Note on the Examination of the Cerebro-spinal Fluid for Arsenic following the Administration of Salvarsan, J. Nerv. & Ment. Dis., No. 12, 1912.
- CANNON, W. G. Bodily Changes in Pain, Hunger, Fear and Rage. New York, D. Appleton & Co., 1915.
- DÉJÉRINE. Etude clinique et anatomo-pathologique sur l'atrophie musculaire des ataxiques, Rev. de méd., 1889.
- La semiologie du système nerveux, Paris, 1901.
- ET EGGER. Contribution à l'étude de la physiologie pathologique de l'incoördination motrice, Rev. neurol., 1903, xi, p. 397.
- ET MARTIN. Séances et memoires de la soc. de biol. June 9, 1889, p. 430.
- DERCUM, F. X. Locomotor Ataxia Occurring in a Blind Tabetic Negro in Whom, though Totally Blind, Closing of the Eyes Immediately Increases Greatly Ataxia of Both Station and Gait. J. Nerv. & Ment. Dis., 1911, xxxviii, p. 281.
- DUCHENNE. Ataxie locomotrice progressive. Arch. gén. d. méd., xii, 1858; *ibid.*, xiii, 1859.
- EDINGER. Der Anteil der Funktion an der Entstehung von Nervenkrankheiten, Wiesbaden, 1908.
- EGGER. La sensibilité osseuse, J. de physiol. et de path. gén., May, 1899.
- Le mécanisme physio-pathologique de l'ataxie du tabés. l'Encéphale, 1901.
- ELSCHNIG. Ueber tabische Sehnervenatrophie, Med. Klin., Berl., 1914, vii, pp. 327-331.
- EPPINGER AND HESS. Vagotonia, translated by Kraus and Jelliffe, New York, 1915.

- ERR, W. Handbuch der Krankheiten des Rückenmarks, 2nd. ed., 1878.
- Zur Aetiologie der Tabes dorsalis, Berl. klin. Wehnsch., 1883; *ibid.*, 1891.
- Die Therapie der Tabes, Samml. klin. Vortr., No. 150, 1896.
- EXNER. Ein neues Operationsverfahren bei tabischen gastrischen Crisen, Deutsche Ztschr. f. Chir., cxi, 1911, p. 576.
- FINGER AND LANDSTEINER. Quoted by Reasoner, loc. cit.
- FÖRSTER, O. Die Physiologie und Pathologie der Koördination, Jena, 1902.
- Zur Symptomatologie der Tabes Dorsalis in preataktischen Stadium und über die Einfluss der Opticusatrophie auf den Gang der Krankheit, Monatschr. f. Psychiat. u. Neurol., Berlin, 1900, vol. viii.
- Resection of the Posterior Nerve Roots of the Spinal Cord, Lancet, 1911, 8, July.
- FOURNIER. De l'ataxie locomotrice d'origine syphilitique, Paris, 1882.
- La période preataxique, Paris, 1885.
- FRENKEL, H. S. Ueber Muskelschlaffheit (Hypotonie) bei der Tabes Dorsalis, Neurol. Centralbl., 1896, No. 8.
- Die Ursachen der Ataxie bei der Tabes Dorsalis, Neurol. Centralbl., 1897, Nos. 15, 16.
- The Treatment of Tabetic Ataxia. English translation by L. Freyberger, London, 1902.
- AND FÖRSTER. Untersuchungen über die Störungen der Sensibilität bei der Tabes Dorsalis, Arch. f. Psychiat., 33.
- FRIEDRIECH. Virchow's Arch., 1863, Bd. 26-27.
- Ueber Ataxie mit besonderer Berücksichtigung der hereditären Formen, *ibid.*, Bd. 68.
- VON FREY, M. Untersuchungen über die Sinnesfunctionen der menschlichen Haut., Abhandl. d. math.-phys. Cl. d. k. sächs. Gesellsch. d. Wissensch., 1896, Bd. xxiii.
- For further references see A Human Experiment in Nerve Division, Rivers and Head, Brain, 1908, vol. xxxi.
- GOLDSCHIEDER, A. Physiologie des Muskelsinnes. Gesammelte Abhandlungen, Leipzig, 1898, vol. ii.

- GOLDSCHIEDER, A. Anleitung zur Uebungs Behandlung der Ataxie. Leipzig, 1899.
- GOWERS. Eye Symptoms in Diseases of the Spinal Cord, *Lancet*, June 16, 1883, p. 1081.
- Syphilis and the Nervous System, London, 1893.
- AND TAYLOR, JAMES. A Manual of Diseases of the Nervous System, London, 1899.
- GROSSMAN, M. Tic or Habit Spasm, *N. Y. Med. J.*, Aug. 14, 1915.
- Chorea: Its Treatment, *N. Y. Med. J.*, May 27, 1916.
- Notes on the Stages of Tabes Dorsalis. Based on the Study of 240 Cases, *Med. Rec.*, Aug. 18, 1917.
- Tabes Dorsalis, *N. Y. Med. J.*, Sept. 1, 1917, p. 402.
- v. GROSZ, E. Ueber tabischen Sehnervenschwund. *Orvosi hetil.* 1897, quoted by Schaffer, *loc. cit.*
- HAUPTMANN AND HÖSSL. *Münch. med. Wehnschr.*, 1910, p. 1581.
- HEAD, RIVERS, AND SHERREN. The Afferent Nervous System from a New Aspect, *Brain*, 1905, vol. xxviii, p. 99.
- AND HOLMES. Sensory Disturbances from Cerebral Lesions, *Brain*, parts ii and iii, p. 102, 1912.
- AND SHERREN. The Consequences of Injury to the Peripheral Nerves in Man, *Brain*, 1905, vol. xxviii, p. 116.
- HENDERSON, D. K. Tabes Dorsalis in Mental Disease. *Rev. Neurol. & Psychiat.*, p. 529 *et seq.*, vol. 9, 1911.
- HELDENBERGH. Cited by Inglerans, *loc. cit.*
- HESS AND EPPINGER. *Vagotonia*, translated by Kraus and Jelliffe, New York, 1915.
- HOLMES, G. A Note on the Condition of the Post-central Cortex in Tabes Dorsalis. *Rev. Neurol. & Psychiat.*, *Edin.*, 1908, vi., pp. 5-11.
- HOMEN. Strang-und Systemerkrankungen des Rückenmarks, *Hand. d. path. Anatomie d. Nervensystems*, 2, 1904.
- HORSLEY AND SLINGER. Upon the Orientation of Points in Space by the Muscular, Arthroidal and Tactile Senses of the Upper Limbs in Normal Individuals and in Blind Persons, *Brain*, 1906, vol. xxix, p. 1.

- HUGHLINGS-JACKSON. The Evolution and Dissolution of the Nervous System, Brit. Med. J., 1884, vol. i, p. 591.
- INGLERANS, LEON. Etude clinique des formes anormales du tabes dorsalis, Thèse de Par., June, 1897.
- JAMES. Text Book of Psychology, London, 1890.
- JENDRASSIK. Ueber die Lokalisation der Tabes dorsalis, Deutsch. Arch. f. klin. Med., xliii, 1888, p. 544.
- Zur Lehre vom Muskeltonus, Neurol. Centbl., No. 17, 1896.
- KNAUER, A. Ueber Pel'sche Augenkrise und einige seltenere Sensibilitätsstörungen bei Tabes dorsalis, Münch. med. Wehnschr., 1908, xiv, pp. 1926-31.
- AND MALONEY. Preliminary Note on the Psychic Action of Mescaline, J. Nerv. & Ment. Dis., 1913, vol. 40, No. 7, p. 425.
- The Cephalograph, *ibid.*, Sept., 1914.
- The Pneumograph, *ibid.*, Feb., 1914.
- KRAFFT-EBBING. Die Aetiologie der progressiven Paralyse. Vort. auf dem internat. med. Kongr., Moscow, 1897.
- LEIMBACH. Statistisches zur Symptomatologie der Tabes Dorsalis. Deutsche Ztschr. f. Nervenhe., vol. 7, Nov. 5, 1895.
- LÉRI. La cécité du tabes, Thèse de Par., 1903-04.
- LEWANDOWSKY. Die Funktionen des zentralen Nervensystems, Berlin, 1907.
- LEYDEN. Die graue Degeneration der Hinterstränge. Virchow's Arch., 1863.
- Zur grauen Degenerationen der hinteren Rückenmarksstränge. Virchow's Arch., 40, 1867.
- AND GOLDSCHIEDER. Die Erkrankungen des Rückenmarks, vol. i, p. 196; vol. ii, p. 589.
- LOWINSKY. Zur Prophylaxe der Tabes Dorsalis, Med. Klin., 1911, No. 35.
- VON MALAISÉ, E. Die Prognose der Tabes Dorsalis. Berlin, 1906.
- MALONEY. The Reckoning Test and Its Uses in Psychiatry. Rev. Neurol. & Psychiat., July, 1911.
- Blindness and Tabes. J. Nerv. & Ment. Dis., vol. 40, No. 9, 1913.

- MALONEY. Fear and Ataxia, *ibid.*, No. 11, 1913.
- The Cure of Ataxia, N. Y. Med. J., Nov. 29, 1913.
- The Mechanism of Mental Processes as Revealed in Reckoning, Psychol. Rev., Baltimore, April, 1914.
- The Determinants of Tabes, N. Y. Med. J., June 20, 1914.
- AND KENNEDY. The Pressure Sense in the Face, Eye and Tongue. Brain, Sept., 1911.
- AND SORAPURE, V. E. Note on Mechanical Support for the Feet in Locomotor Ataxia, Med. Rec., May, 1914.
- Relief of States of Vascular, Muscular and Mental Tension, N. Y. Med. J., May, 1914.
- MARIE, PIERRE. Leçons sur les maladies de la moelle, Paris, 1892.
- Tabes, Traité de Médecine de Charcot et Bouchard, Paris, 1895.
- MARINESCO. De la topographie des troubles sensitifs dans le tabès; ses rapports avec les sensations des tabétiques, Semaine méd., 1907.
- Contribution à l'étude de l'histologie et de la pathogenie du tabès, *ibid.*, 1906.
- MARTIN, JOANNES. Del' atrophie du nerf optique et de la valeur pronostique dans la sclérose des cordons postérieures de la moelle, Thèse au Berne, 1890.
- MATSUNAGA. Über Tabes und Aortenerkrankungen, Inaug. Dissert., Munich, 1910.
- MCDONAGH, J. E. R. Biology and Treatment of Venereal Diseases, London, 1915.
- MCDUGALL, W. The Nature of Inhibitory Processes within the Nervous System, Brain, 1903, vol. xxvi, p. 153.
- MCINTOSH, J., FILDES, P., HEAD, H., AND FEARNSIDES, E. G. Parasyphilis of the Nervous System, Brain, July, 1913, part 1, vol. 36.
- MENDEL, E. Die Tabes beim weiblichen Geschlecht, Neurol. Centbl., 1901, No. 1.
- MENDEL, KURT. Zur Paralyse-Tabes-Syphilisfrage, Neurol. Centbl., 1905, No. 1.

- MENDEL, KURT AND TOBIAS, ERNST. Die Tabes der Frauen, Berlin, 1912, p. 48.
- MEYER, OTTO. Beitrag zur Kenntnis der nicht paralytischen Psychosen bei Tabes dorsalis, Monatschr. f. Psychiat. u. Neurol., No. 3.
- MINGAZINNI AND SALGADORRI. Considerazione cliniche sulla tabe ereditaria, Riv. di patol. nerv., ii.
- MIRALLIÉ ET DESCLAUX. De l'état des nerfs oculomoteurs dans l'hémiplégie organique de l'adulte. Rev. Neurol., 1903, ii, p. 649.
- MOEBIUS. Ueber die Tabes, Berlin, 1897.
- MOTT, F. W. Tabes in Asylum and Hospital Practice, Arch. Neurol. London Country Asylums, 2, 1903.
- NAGEOTTE, J. Regenerations collaterales des fibres nerveuses, etc., lésions tabétiques des racines médullaires, Nouv. Iconog. de la Salpêtrière, 1906.
- NICHOLS, H. J. Jour. Exp. Med., 1914, xix, No. 2.
- AND HOUGH, W. H. Demonstration of Spirochaeta Pallida in the Cerebrospinal Fluid, J. Am. Med. Ass., Jan. 11, 1913, p. 108.
- NOEHTE. Die Behandlung der Tabes, speziell ihrer rudimentären Form, und deren Beziehungen zu psychopathischen Störungen. Deutsche med. Wchschr., Berl., 1913, xxxix, p. 999-1002.
- NOGUCHI. Experimental Research in Syphilis with Special Reference to Spirochaeta pallida, J. Am. Ass., April 20, 1912.
- The Direct Cultivation of Treponema pallidum Pathogenic for the Monkey, J. Exp. Med., 1912, xv, No. 2.
- J. Exp. Med., 1913, p. 232.
- Presse méd., 1913, viii, p. 805.
- The Transmission of Treponema pallidum from the Brains of Paralytics to the Rabbit, J. Am. Med. Ass., July 12, 1913.
- NONNE. Anatomische Untersuchungen von Zehn Fällen von Tabes dorsalis mit besonderer Berücksichtigung des Verhaltens der peripheren Nerven, Jahr. d. Hamburger Staatskranken Anstalten, I, 1889.
- Syphilis und Nervensystem, Berlin, 2 ed., 1916.

- NONNE AND HOLTZMANN, W. Ueber Wassermann Reaktion in Liquor spinalis bei Tabes dorsalis sowie ueber quantitative Auswertung von Stärkegraden der W. Reaktion bei syphiligen Krankheiten des zentral Nervensystems, Monatschr. f. Psych. u. Neurol., Berlin, 1910, xxvii, 128-152.
- OPPENHEIM. Zur pathologischen Anatomie der Tabes dorsalis, Berl. klin. Wehnschr., 1894.
- Lehrbuch der Nervenkrankheiten, Berlin, 1908.
- ORR. A Contribution to our Knowledge of the Course of the Lymph Stream in the Spinal Roots and Cord, Rev. Neurol. & Psychiat., Edinburgh, 1903, vol. i, p. 639.
- AND ROWS. Toxic Infection of the Central Nervous System, Rev. Neurol. & Psychiat., Edinburgh, 1907, vol. v, p. 345.
- PAZ, DE LA. *See* Cannon, *loc. cit.*
- RAYMOND. Leçons sur les maladies du système nerveux, Paris, 1897, vol. II, p. 574.
- REASONER, MATHEW A. Some Phases of Experimental Syphilis, J. Am. Med. Ass., Dec. 16, 1916, vol. lxvii, pp. 1799-1805.
- RIVERS AND HEAD. A Human Experiment in Nerve Division, Brain, 1908, vol. xxxi, p. 323.
- ROMBERG. Lehrbuch der Nervenkrankheiten, quoted by Sir David Ferrier, Tabes Dorsalis, Lancet, March 31, 1906, p. 881; *ibid.*, April 7, p. 951.
- ROSENTHAL. Cited by Inglerans, *loc. cit.*
- RYDLEWSKI. Über Psychosen bei Tabes dorsalis, Inaug.-Dissert. Greifswald, 1909.
- SARBO, VON. Klinische und Stätistische Daten zur Symptomatologie der Tabes, Deutsch, Zeit. f. Neurol., Vol. 23, 1913.
- SCHAEFFER, K. Das Verhalten der Spinal Ganglienzellen bei Tabes auf Grund Nissl's Farbung, Neurol. Centralbl., 1898.
- Article on Tabes Dorsalis, Lewandowsky's Handbuch der Neurologie, Berlin, 1911, 2nd vol., p. 957.
- SCHÜPFER, FERRUCIO. Sur valore prognostico del l'atrofia dei nervi ottici, Riv. sper. di freniat., Jan., 1901, p. 249 *et seq.*
- SHUSTER, PAUL. Die abortiven Formen der Tabes Dorsalis und der übrigen syphiligen Nervenkrankheiten, Med. Klin., 1913, Nr. 18.

- SHERINGTON, C. S. The Integrative Action of the Nervous System, New Haven, Conn., 1911.
- SLINGER AND HORSLEY. Upon the Orientation of Points in Space by the Muscular Arthroidal and Tactile Senses of the Upper Limbs in Normal Individuals and in Blind Persons, *Brain*, p. 1 *et seq.*, 1906, part 1.
- SPILLER, W. G. The Amaurotic Form of Tabes Dorsalis—Tabes Arrested by Blindness, *Phila. Med. J.*, Jan. 29, 1898.
- Tabetic Ocular Crises, *J. Am. Med. Ass.*, March 18, 1916.
- SPILLMANN, P., AND PERRIN, M. Le rôle de la syphilis dans l'étiologie du tabes dorsalis, *J. méd. franç.*, Paris, 1909, iii, pp. 642-649.
- STARGARDT, K. Ueber die Aetiologie der tabischen Arthropathien, *Arch. Psychiat.*, Berlin, 1912, xlix, 936-954.
- Ueber die Ursachen der Sehnervenschwunde bei Tabes und der progressiven Paralyse, *ibid.*, 1913, ii, pp. 711-976, 4 plates.
- STRÜMPPELL, A. Ueber die Vereinigung der Tabes dorsalis mit Erkrankungen des Herzens und der Gefässe. *Deutsche med. Wehnschr.*, No. 47, 1907.
- TOMASCZEWSKI. Ueber die Ergebnisse der Superinfektion bei der Syphilis der Kaninchen, *Berl. klin. Wehnschr.* No. 31, p. 1447.
- TROUSSEAU. Cited by Inglerans, *loc. cit.*
- VEDDER, EDWARD BRIGHT. The Prevalence of Syphilis in the Army, *War Dept. Bull.*, No. 8, June, 1915.
- VULPIAN. Cited by Inglerans, *loc. cit.*
- WASSERMANN. For references see Nonne's Syphilis und Nervensystems.
- WEILER, KARL. Untersuchung des Kniesehnen-reflexes beim Menschen, *Ztschr. f. d. ges. Neurol. u. Psych.* Bd. 1, H. 1, p. 116, 1910.
- WESTPHAL, C. *Arch. f. Psychiat.*, xvii, 547.
- WARTHIN, ALFRED SCOTT. *Am. Jour. Med. Sciences*, Vol. clii, p. 508, 1916.
- WILE, UDO J., AND STOKES, JOHN H. Further Studies on the Spinal Fluid with Reference to the Involvement of the

Nervous System in Early Syphilis. Jour. Am. Med. Ass., May 1, 1915.

WILLCOX. Quoted by McIntosh, Fildes, etc., *loc. cit.*

WILSON, S. A. K. The Pathology of Two Cases of Tabetic Amyotrophy. Rev. Neurol. and Psych., Edinburgh, Aug., 1911, pp. 401-418.

WOLF, H. F. The Symptom Ataxia, Its Successful Treatment. N. Y. Med. J., Sept. 9, 1916.

— The Treatment of Locomotor Ataxia by the Maloney Method, *ibid.*, July 21, 1917.

INDEX

- Abducens lesions in tabes, 13
- Abrahamson and Pollen, treatment of spasmodic muscular affections, 273
- Accessibility of tissues to spirochete, 5
- Achilles jerk, 33
- Acquired immunity to spirochetes, 138-139
- Acute ataxia, 72, 73
- Adrenalin, action of, 136
 - effect of, 136
 - use of, to prevent lesions after salvarsan, 146
- Age, at death in tabes, 163, 164
 - at onset of tabes, 163
- Aids to stability, mechanical, 240-241
- Algesimeter, 48
- Algometer, 48
- "All fours" posture, 249
- Alternative reflex paths, 25, 26
- Amyl alcohol, site of lesions, 14
- Analgesia, 52, 53
- Analgesics, 218
- Anatomical basis of tabetic signs, 152
- Ankle exercises, 255
- Anterior horn cell lesions in tabes, 13
- Antisyphilitic substances, 166
- Anxiety neurosis in tabes, 177
- Argyll-Robertson pupil, 119, 153
- Arm jerks, 33
- Arsenic, 166, 170, 172
- Arsenical preparations, 172
- Arthropathies, 137
- Asynergia, 44
- Ataxia, acute, 72, 73
 - amount of improvement in, following blindness, 103
 - an emotional expression, 82
 - and blindness, relation of, 104
 - and mental state, 77
 - and postural loss, 70
 - and reflex loss, 70
 - and sensory loss, 70
 - and tabetic lesions, 73, 74
 - blindfold treatment, advantages of, 272-273
 - co-efficient of, 78
 - course of, 71, 72
 - definition of, 69
 - disappearing in blindness,
 - Abrahamson's case, 98, 99
 - Benedikt's case, 97
 - Benenati's cases, 98
 - Bouchaud's case, 97
 - Förster's cases, 98
 - Inglersans' cases, 97, 98
 - Léri's case, 99
 - Martin's case, 97
 - duration of treatment of, 261-262
 - extent of improvement, 260, 261
 - following blindness, 101
 - Frenkel's method of treatment, 271-272
 - index of mental state, 186
 - liability to, 78
 - mainly a spatial defect, 81
 - mechanism of, 83
 - onset of, 71
 - palliation of, 229

- Ataxia, permanency of cure, 262
 preceding blindness, 96-104
 preliminary treatment of,
 a. mental, 244-247
 b. muscular, 245
 prevention of, 212, 228
 recording of, 228
 rôle of the several parts of the
 muscular apparatus in, 69
 theories of, 69
 treatment in blindness, 261
 variations in, 74
 Ataxic attitude, 229
 complexes, 77
 foot, mechanics of, 230-231
 moment, 73
 threshold, 78
 Atropin in treatment of vago-
 tonia, 191
 Attacking force of persisting
 spirochete, 11
 Attention, rôle in sensory percep-
 tion, 47, 48, 49
 Attitude. *See* posture
 Auditory nerve. *See* cochlear
- Back braces, 240-241
 Baths, 204
 Beaussart's nerve spirochete se-
 ries, 9
 Bedridden tabes, determining fac-
 tors in, 161, 162
 Belière's nerve spirochete series,
 8
 Benedikt's "law," 94
 advocates of, 95
 Déjerine and Martin on, 95
 opponents of, 95
 Blind tabetics, postural sense of,
 59
 sensory perception in, 59
 Blindfold movement, 247
 Blindfolding, mask for, 247
 to focus attention, 247
- Blindness, amount of improve-
 ment of ataxia in, 103
 and ataxia, 96-104
 and ataxia developing simulta-
 neously, 101
 and ataxia, relations of, 104
 and deafness, 105
 and pain, 105
 effect of drugs on, mercury, 94
 mescaline, 89
 salvarsan, 94
 following ataxia, 9
 frequency of, 91, 94
 improvement in, 93, 94
 influence upon ataxia, 101
 influence upon mental state, 102
 preceding ataxia, 92
 rate of development, 92
 treatment of, 223
 Boot, tabetic, 238
 Braces, back, 240-241
 foot, 237
 knee, 240-241
 Breathing exercises, 196
 Burr, cause of death in tabetics, 11
- Cardiac diseases in tabes, 164
 Cardiovascular and nervous syph-
 ilis, 11
 Causal relation of syphilis to
 tabes, 2
 Causes of death in tabes, 11, 164
 Causes of imperfect defence, 149
 Central nervous system, determi-
 nants of invasion, 12
 Centers of vegetative activity, 119
 Cephalograph, 205
 Cephalograph, measurement of
 swaying, 65
 Cerebellum lesions in tabes, 13
 functions of, 30, 31
 Cerebral theory of ataxia, 69, 71
 Cerebrospinal fluid in tabes, 152
 Charcot joints. *See* arthropathies

- Chemical action and defense, 145
- Cocain in gastric crises, 220
- Cochlear nerve, 113
- Complex movements, training in, 255
- Complexes, ataxic, 77
- Complexes, fear, 177
- Constitutional influences in nerve syphilis, 12
- Cortical lesions in tabes, 12, 13
- Cortical motor reactions, 42
- Course of ataxia, 71, 72
- Course of optic atrophy, 87
- Creeping, 257
- Creeping, knee-guards for, 256
- Crises, bladder, 129
 - duration of, 131
 - gastric, 127
 - genital, 130
 - ocular, 127
 - pain, 127
 - rectal, 129
- Crises and mental state, 130-131
- Cystitis in tabes, 164
- Deafness and mental state, 114-115
- Deafness, daily variations in, 115
 - detection, 113
 - psychic, 113
 - tabetic, 113
 - treatment of, 224
- Death in tabes, 163, 164
 - causes of, 11, 164
- Death rate among tabetics, 164
- Deep reflexes, 32, 33
- Defense, causes of imperfection, 149
 - effect of mental strain upon, 148-149
 - mechanism of, 136
 - substances, existence of, 144-145
- Déjerine, frequency of root lesions in paresis, 12
- Delayed perception from incomplete lesions, 52
- Depression in tabes, 178
- Determinants of invasion of central nervous system, 12
 - of site of initial invasion, 15
 - of spread of invasion, 16
- Development of ataxia, mechanism of, 80
- Diagnosis of tabes, 153
- Diagnosis, precipitant of ataxia, 194
- Diplopia, cure of, 207
 - examination of, 107
 - frequency, mechanism, causes of, 105
 - treatment of, 215
- Direction, training in, 251
- Direction of movement, threshold of, 58
- Discovery of tabes, 1
- Dislocations, 35
- Distribution of sensory loss, 52, 53
 - of spirochete, 13, 14
 - of postural loss, 58
- Dosage of exercises, 207
 - of movement exercises, 248
 - of postural exercises, 256
- Drainage theory of reflex action, 28
- Drug treatment, physical factors in, 174
 - psychological factors in, 174
- Duchenne, discovery of tabes, 1
 - on diagnosis of tabes, 155
- Duration of pretabetic period, 156-158
- Edinger's fatigue theory, 14, 15
- Effort and movement, 76
- Eighth nerve. *See* vestibular and cochlear
- Electricity, 251
- Equilibration, center of, 31

- Erb's nerve spirochete series, 9
 Ergot, site of lesions, 14
 Exciting cause of tabes, 2
 Exciting organism of tabes, 5
 Exercises, ankle, 255
 breathing, 196
 eye, 206
 graduation of, 207
 head, 205
 head and neck, 253
 hip, 254
 knee, 254
 passive, 198-199
 resistance, 251
 rest, 196-204
 trunk, 253
 Exner's operation for gastric
 crises, 221
 Eye exercises, 206

 Facial lesions. *See* seventh
 Factors determining response to
 stimuli, 49
 Factors determining fate of sen-
 sory images, 48
 Factors in a voluntary movement,
 76
 Factors in posture, 62
 Factors in pretabetic period, 156
 Factors of muscle tone, 31
 Fatigue in tabes, 176
 Fatigue theory, Edinger, 14, 15
 Fear and movement, 68
 and posture, 68
 complexes, 177
 in tabes, 176, 177
 Feet, tabetic, 229, 230
 Fifth nerve lesions, 110
 Flat foot, 34, 229, 230
 Foot, aims in bracing, 237
 Foot blisters, 231
 Foot, bracing, 237
 Foot cast, of foot in action, 232
 of foot at rest, 232

 Foot, structural changes in tabetic,
 236
 Förster's operation for gastric
 crises, 220-221
 Frenkel's method, 272
 Frequency of blindness, 91, 92
 of muscular atrophy, 17
 of optic atrophy in tabes, 12
 of paresis following optic
 atrophy, 12
 of peripheral neuritis in tabes,
 12
 of posterior root lesions in optic
 atrophy, 12
 Frequency of tabetic signs and
 symptoms, Abadie, 151
 Leimbach, 151
 Sarbo, 151
 Schaffer, 151
 Function, cerebellar, 30, 31
 Function of muscle spindle, 24

 Gastric crises, cocain in, 220
 measurement of, 218
 mental treatment of, 219
 morphin in, 220
 nature, 127
 novococain in, 220
 palliative treatment of, 219
 prevention of, 219
 rest in, 219
 root section in, 220-221
 General paresis following optic
 atrophy, 12
 General paresis, frequency of root
 lesions in, 12
 Genu recurvatum, 34
 Glands, endocrinous or internal
 secreting, 135
 Goldscheider's instrument for
 measuring posture, 57
 Gordon Holmes, cortical lesions in
 tabes, 12, 13
 Grossman, cure of ataxia, 274

- Grossman, treatment of chorea, 273
- Hallucinatory conditions in tabes, 178
- Head exercises, 205
and neck exercises, 253
- Hearing, mechanism of, 112
- Henderson, psychoses of tabes, 178
- Hip exercises, 254
- Hitzig's accident theory, 15
- Hormones, nature and action, 135
- Horsley and Slinger, postural sense in blind, 59
- Hypochondria in tabes, 178
- Hypotonia, effect on joints, 34
result of muscle stretching, 34
- Immunity of tissues to spirochetes, 132-139
- Imperfect sensation from incomplete lesions, 52
- Incubation period. *See* pretabetic period
- Infection general in syphilis, 5
- Influence of spirochete's distribution, 14
optic on spinal tabes, 16, 17
nerve lymphatics upon spirochete's distribution, 14
- Instruments for measuring stimuli, 48
- Integration, spinal, 28
- Internal secreting glands, 135
- Involuntary micturition, treatment of, 222-223
- Iodids, 170
- Isolation in treatment, 210
- Isolation of tabes, 177
- James' theory, 195
- Jaw jerk, 33
- Jendrassik, cortical lesions in tabes, 12
- Joint braces, 240-241
- Knee, braces, 240-241
exercises, 254
- Knee-guards for creeping, 256
- Knee-jerk, a reflex act, 32
briskness of, 32
eliciting, 32
extent of, 32
fatiguability of, 32
influence of flexor tone of, 32
nature of, 32
normal variations of, 32
pathological changes of, 33
reënfacement of, 32
- Kneeling, 249, 258
- Law of relays, 39
- Law relating blindness and ataxia, 104
- Lesions of basal nuclei in tabes, 13
- Lesions. *See* tabetic lesions
- Lesions, sequence of, 16
- Lethality of tabes, 164
- Liability of neuron to invasion, 15
- Liability to syphilis of central nervous system, 12
- Light perception, measurement of, 89
- Lightning pains, 126. *See* pains, lightning
- Loss of pinprick pain, 52
of postural sense, 58
of pressure pain, 124-125
of tactile sense, 53
of temperature sense, 53
of vibration sense, 54
of vision, course of, 88
of vision, degree, 88
of vision, measurement of, 88
- Lymphatics, influence upon site of attack, 14
- Lymphatics in optic nerve, 13
- Lymphatics in posterior root, 13
- Macdougall's drainage theory, 28
- Maintenance of posture, 62

- Manic depressive insanity in tabes,** 178
Marie, cause of posterior root lesion, 19, 20
Mask for blindfolding, 247
Masking of reflex defect by cortical action, 43
Massage, 257
Matsunaga, cardiac disease among tabetics, 11
Measurement of light perception, 89
 of loss of vision, 88
 of mental state, 179
 of postural loss, 56
 of swaying, cephalograph, 65
Mechanical aids to stability, 240-241
Mechanism of hearing, 112
 of micturition, 131-132
 of pupillary movement, 118
 of Rombergism, 57
 of suppression of postural images, 57, 58
 of voluntary movement, 42
Melancholia in tabes, 178
Mental factor in muscular weakness, 37
Mental training through movement, 204-207
Mental state and ataxia, 77
 and crises, 128
 and deafness, 114-115
 and movement, 76
 and pain, 130-131
 and virility, 130
 deterioration of, 183-184
 effect of symptoms on, 184
 influence of posture upon, 62
 influence upon signs of, 185
 influence upon symptoms of, 184
 measurement of, 179
 principles of treatment of, 188
Mental state, relief through treatment of eye symptoms of, 215
Mental strain, effect on defense, 148-149
Mental stress and tabes, 146-147
Mercury, 166, 170
 failure of, in treatment of optic atrophy, 169
 modes of administration of, ingestion, 170
 inhalation, 171
 injection, 171
 inunction, 170-171
Meyer, psychoses of tabes, 178
Micturition, involuntary, mechanism of, 133
 voluntary, mechanism of, 131-132
Mobility of joints, 34, 35
Morel Lavallée's nerve spirochete series, 9
Morphin in gastric crises, 220
Motor nerve lesions in tabes, 13
Motor reactions, cortical, 42
 reflex, 43
Movement, after-images of, 79
 and effort, 76
 and mental state, 76
 and posture, 248
 critique of, 79
Movement exercises in, recumbent posture, 248
 "all fours," 249
 kneeling, 249
 standing, 249
Movement exercises, order of using, 252
 pulse in, 259
Movement, factors in perfection of, 75
Movement in fear, 68
Movement, preliminary stabilising of, 248-249

- Movement, rôle of vision in learning, 75
 Movement, threshold of, 57
 Movements, complex, training in, 455
 Movements, creeping, 257
 kneeling, 258
 simple, 251
 cardinal qualities of, 251
 training in strength, 251
 walking, 258
 Moving by visual direction, 245
 Muscle spindle, 23
 tone center, 31
 Muscular apparatus, sensory receptors, 54, 55
 Muscular atrophy, frequency of, 17
 Muscular weakness, factors in, 37
 Myelin sheath, degeneration of, 52
 function of, 52

 Nageotte, tabetic posterior root lesion, 21
 Nature of posterior root lesions, 21
 Nerve lymphatics, spirochete in, 13
 Nerve relays, cerebellar, 30
 sentient, 45, 47
 Nerve spirochete series, Beausart's, 9
 Belière's, 8
 Erb's, 9
 Morel Lavallée's, 9
 Nervous lesions, specific spirochete, 5
 Neurasthenia, syphilitic, 153
 treatment of, 190, 191, 192
 Noguchi, discovery of spirochete in tabes, 4
 Nonne, peripheral neuritis in tabes, 12
 Normal swaying, 62, 63

 Normal and morbid swaying, differentiation, 64, 65
 Nourishing the tabetic, 203
 Novocain in gastric crises, 220
 Nucleinic acid, 175

 Occupation, adjustment of tabetic's, 211-212
 Occupational incidence of tabes, 147
 Occupational influences in nerve syphilis, 12
 Occurrence of syphilis in tabes, 2
 Oculomotor lesions in tabes, 13
 Onset of ataxia, 71
 Opiates. *See* morphin
 Oppenheim, peripheral neuritis in tabes, 12
 Optic atrophy, 87
 course of, 87
 effect on spinal tabes, 16, 17
 frequency of posterior root lesions in, 12
 nature, 87
 ophthalmoscopic findings, 87
 results of, 88
 treatment of, 223
 Optic atrophy and diplopia, 107
 Optic atrophy followed by general paresis, frequency of, 12
 Optic nerve, atrophy of, 87
 lymphatics, 13
 site of amyl alcohol lesions, 14
 Optic tabes, cause of abortive nature of, 150
 influence on spinal, 16, 17
 nature of, 87
 Orr and Rows, posterior root lymphatics, 13

 Pain, an emotional outlet, 217
 and mental state, 216
 Pain crisis, treatment of, 218
 Pain in treatment of, 217

- Pain, lightning, distribution of, 126
 cause of, 126
 nature of, 127
 occurrence of, 126
 onset of, 126
- Pain, measurement of, 216
- Pain pressure, relation to vegetative nervous system, 120-124
- Pain pinprick, loss of, 52, 53
- Pain, reaction to, 216
- Pain, subacute, treatment of, 218
- Pain, treatment of, 217-218
- "Paralytic" period, 161
 nature of, 183-184
- Paranoia in tabes, 178
- Paresis. *See* general paresis
- Paresthesia, 53
- Passive muscular exercises, 198-199
- Peripheral neuritis in tabes, frequency of, 12
- Persisting spirochete, types of, 11
 attacking force of, 11
- Plaster casts of foot, 232
- Pneumograph, 204
- Pneumonia in tabes, 164
- Poirier, lymphatics in optic nerve, 13
- Posterior column fibers, cerebellar, 29
 medullary, 29
 spinal, 29
- Posterior nerve root, central
 process, course of, 22
 situation of, 18
 structure of, 18
- Posterior root coverings, 18
 ganglion, 18
 ganglion cells, 18, 19
- Posterior root lesion, anatomical
 result of, 39
 and meningitis, 20
- Posterior root lesion, and spinal
 ganglion cell, 19, 20
 effect on radiation of, 39
 in optic atrophy, frequency of, 12
 in paresis, frequency of, 12
 physiological result of, 39
 primary, 21
 reflex disintegration of, 39
 results, anatomical, 22
 results of complete, 51
 results of incomplete, 51, 52
 spread of, 21
- Posterior root lymphatics, 13
- Posterior root section in gastric
 crises, 220-221
- Posterior roots, site of ergot
 lesions, 14
- Postural aids, 65
- Postural appreciation, effect of
 lesions on, 56
- Postural deterioration, 65, 66
- Postural exercises, 249
 dosage of, 250
- Postural images, peripheral after-
 images of movement, 55, 79
- Postural loss and ataxia, 70
- Postural loss, distribution of, 58
 measurement of, 56
- Postural sense, normal blind, 59
 tabetic blind, 59
- Postural suppression and fear, 68
 and visual substitution, 67
- Postural threshold, normal varia-
 tions of, 57
- Posture and fear, 68
- Posture, factors in, 62
 influence of mental state, 62
 its acquirement, 61
 its maintenance, 62
 sensation of, 55
- Pre-ataxic period, 159
 duration of, Fournier, 159
 Grossman, 160

- Pre-ataxic stage, duration of, 77
- Predilection, cultural, 16
- Pressure pain loss, 124-125
 - distribution of, 125
- Pressure pain, relation to the vegetative nervous system, 120-124
- Pressure sense, relation to vegetative nervous system, 120-124
- Pretabetic period, duration of, Grossman, 157
 - Lowinsky, 157
- Pretabetic period, effect of drugs on duration of, 167
- Prevention of Rombergism, 205
- Principles of psychological treatment, 188
- Principles of treating mental state, 188
- Progression, training in, 256
- Properties of reflex action, 27
- Protected tissues, syphilitic invasion, 11
- Psychological treatment, principles of, 188
- Psychoses of tabes, 178
- Psychotherapy, principles of, 188
- Pupil, Argyll-Robertson, 119
 - changes in tabes, 118, 119
 - mechanism of movement, 118
- Racial incidence of tabes, 147
- Radiation, 27
- Reciprocal relations of mental state and symptoms, 185
- Reckoning test in tabes, 177-182
 - mode of use, 180-181
- Recumbent posture, 249
- Reflex action, agonists and antagonists, 28
 - properties, 27
 - reflex arc, afferent path, 24
 - synergic, 28
- Reflex collateral, course, 22
- Reflex defect during voluntary movement, 44
- Reflex disintegration, difficulty of measurement, 42
 - initial and ultimate results, 39
 - rapidity of development of lesions, 39
 - site of lesion, 40
- Reflex loss and ataxia, 70
- Reflex theory of ataxia, 69
- Reflexes, deep, 32, 33
 - tendon, 32, 33
- Reënforcement of reflex by cortical action, 43
- Relation of antisyphilitic treatment to tabes, 168
- Relation of meningitis to posterior root lesion, 20
- Relation of spinal ganglion cell to posterior root lesion, 19, 20
- Relay, law of, 39
- Remissions in tabes, 159
- Renal diseases in tabes, 164
- Resistance exercises, 251
- Resistance in degenerated nerve, 52
- Resistance of nerve cells, variations in, 14
- Resistance in synapse of degenerated nerve, 52
- Resistance, synapse, seat of, 27
- Rest exercises, 196-204
- Rest exercises, effect, on blood pressure, 200-201
 - on pulse, 201
 - on musculature, 200
 - on mental state, 200, 202
- Rest exercises, physiological effects of, 200
 - routine procedure in, 202
- Romberg, recognition of tabes, 1
- Rombergism, diagnosis of, 64
 - mechanism of, 57, 58

- Rombergism, measurement of, 65, 226
 prevention of, 205-227
- Romberg's sign, 63
 analysis, 227
 eliciting of, 63, 64
 first motor disability, 226
- Root lesions and aortic disease, 11
- Root lesions. *See* posterior and anterior root lesions
- Rydlowski, psychoses of tabes, 178
- Salvarsan, failure in treatment of optic atrophy, 169
 followed by fulminating spread of lesions, 146
 intraspinal administration, 173
- Salvarsanised serum, 173
- Secondary lesions, specific spirochete, 5
- Senile tabes, 146
- Sensation, complexity of simplest, 50
- Sense of pressure, relation to the vegetative nervous system, 120-124
- Sensory images, fate of, 47, 48
 specificity of, 47
- Sensory impulses, delayed by degeneration, 52
 speed of, 52
 specific, 45
- Sensory loss and ataxia, 70
 distribution of, 52, 53
- Sensory nerve of voluntary muscle, origin, course, termination of, 22
- Sensory receptors, 24
 specificity of, 45
 of muscular apparatus, 54, 55
- Sensory theory of ataxia, 69
- Sensory threshold, estimation of, 48, 49
 variations of, 48, 49
- Sequence of lesions, 16
- Seventh nerve lesions, 110, 122
- Sex incidence of tabes, 147
- Shock as a cause of tabes, 146
- Shoe, tabetic, 238
- Signs, influence of mental state on, 185
- Sign Romberg, 63
- Simple reflex arc, mechanism of, 24
- Site of initial attack, determinants of, 15
- Sites attacked in tabes, 12
- Sites of syphilis in central nervous system, 12
- Sitting posture, 249
- Slinger and Horsley, postural sense in the blind, 59
- Specific strains of spirochetes, 5
- Spinal integration, 28
- Spinal tabes, cause of progressive nature of, 149
- Spinal vegetative fibers, 119
- Spirochete, accessibility of tissues, 5
 distribution, 13, 14
 exciting organism of tabes, 5
 in lymphatic channels of nerves, 13
 in tabes, Noguchi's discovery, 4
 of nervous lesions, 5
 of secondary lesions, 5
 of tertiary lesions, 5
 persisting, 11
 specific strains, 5
- Spread of invasion, determinants of, 16
- Squint. *See* strabismus
- Stability, mechanical aids to, 240-241
- Standing posture, 249
- Stargardt on optic tabes, 87
- Stargardt, posterior root lesion parenchymatous, 21

- Strabismus, 107-109
 cure of, 207
- Strümpell, aortic disease and root lesions, 11
- Suppression of postural images, 57, 58
 of visual images, 107
- Swaying, measurement of, cephalograph, 65
 normal, 62, 63
- Sympathetic fibers, 117, 118
 lesions in tabes, 13
- Symptomatic treatment, psychotherapeutic, 188
- Symptoms and signs of tabes, 151
- Symptoms, effect on mental state, 184
 measurement and analysis of, 215
 of fatigue, 176
 of fear, 176-177
 pre-ataxic, 215
 prevention of, 212-214
- Synapse, cells, changes in tabes, 24
- Synergia, center of, 31
- Synergic action, 28
- Syphilis a general infection, 5
 causal relation to tabes, 2
 in central nervous system, sites of, 12
- Syphilis of central nervous system, constitutional influences of, 12
 liability to, 12
 occupational influences of, 12
 spread of, abortive, 159
 fulminating, 156, 158
 tardy, 157, 158
 environmental influences affecting, 12
- Syphilis, prosecution for, 192-193
- Syphilitic invasion of protected tissues, 11
- Syphilitic neurasthenia, 153
 treatment of, 190, 191, 192
- Tabes, abducens lesions, 13
 age at death, 163
 age at onset, 162, 163
 and thyroid gland, 146
 anterior horn cell lesions, 13
 causes of death in, 164
 cerebellar lesions, 13
 cerebrospinal fluid in, 152
 cortical lesions, 12, 13
 diagnosis of, 153
 discovery of, 1
 motor nerve lesions, 13
 occupational incidence, 147
 occurrence of syphilis in, 2
 oculomotor lesions in, 13
 producing depression, 177
 producing fatigue, 176
 pupillary changes in, 118, 119
 racial incidence of, 147
 senile, 146
 sex incidence of, 147
 sites attacked, 12
 sympathetic lesions of, 13
 symptoms and signs of, 151
 theories of causation of, 2
 types of, 162
 vagus lesions, 13
 vestibular lesions, 13
 Wassermann reaction in, 152
- Tabetic boot, 238
 deafness, course of, 114-115
 incipient, treatment of, 190-192
- Tabetic lesions, affecting micturition, 132
 and ataxia, 73, 74
 anterior horn cell, frequency, result of, 37
 auditory, 13
 cerebellar, 13
 cochlear nerve, extent of, 114
 result of, 114

- Tabetic lesions, compatible with
 syphilitic origin, 4
 cortical, 12, 13
 cranial nerves, 86
 eighth nerve. *See* vestibular
 and cochlear
 fifth nerve, 110
 inflammatory, not toxic, 4
 motor nerve, 13
 motor nerve, results of, 37
 oculomotor, 13
 olfactory, 87
 optic, 12, 87
 peripheral nerves, 12, 13
 peripheral processes of spinal
 ganglion cell, 19
 posterior root, meninges, 19
 seventh, 110
 spinal ganglion cells, 18, 19
 sympathetic, 13
 vagal, 13
 vestibular, 13
 Tabetic, potential treatment of,
 190-192
 Tabetic, pre-ataxic treatment of,
 194
 Tabetic psychosis, 178
 shoe, 238
 signs, anatomical basis of, 152
 symptoms, interdependence, 186
 Tactile sensibility loss, 53
 Temperature, loss, 53
 Tendon reflexes, 32, 33
 Theories of ataxia, 69
 Theory, drainage, 28
 James, 195
 Threshold of ataxia, 78
 of change of illumination, 90
 of direction of movement, 58
 of light perception, 90
 of movement, 57
 Thyroid gland in tabes, 146
 Tissue immunity to spirochetes,
 138, 139
- Toe clutching, 230
 Toes, callous formation on, 231
 Training in cerebral control, 204,
 205, 206
 in direction, 251, 252
 in extent, 251, 252
 in mental capacity, 207
 in rhythm, 251
 Treatment by drugs, 166
 curative, 166
 preventive, 166
 Treatment of arthropathies, 261
 of blindness, 223
 of deafness, 224
 of incipient tabes, 190-192
 of involuntary micturition, 222-
 223
 of optic atrophy, 223
 of syphilitic neurasthenia, 190-
 192
 Trigeminal lesions. *See* fifth
 Trunk exercises, 253
 Tuberculosis in tabes, 164
 Types of persisting spirochetes,
 11
- Urination. *See* micturition
- Vagal fibers, 117, 118
 irritation and gastric crises, 128
 Vagotonia, precursor of tabes, 153
 treatment of, 190-191
 Vagus lesions in tabes, 13
 Vascular disease in tabes, 164
 Vegetative nervous system, affer-
 ent and efferent spinal fi-
 bers, 119
 chemical stimulation to defense,
 145
 composition of, 117
 development of, 117
 Vegetative nervous system lesions,
 arthropathies, 137, 138
 atrophies, 137

- Vegetative nervous system lesions,
 - effect on defense, 150
 - fractures, 137
 - infections, 137
 - trophic ulcers, 137
- Vegetative nervous system, mental
 - control of, 150
 - plexuses, 119
 - relation of spinal afferent to posterior root ganglion, 120-124
 - sensory function of, 120-124
- Vegetative nervous system, spino-medullary centers, 119
- and metabolism, 136
- Vestibular impulses, origin, distribution of, 29
- lesions in tabes, 13
- Vestibular lesions, detection of, 111
- Vestibular organ of tone, situation, receptors of, 29, 30
- Vibration, loss of, 54
- Virility and mental state, 130
- Visual criticism of movement, 80
- Visual images, detection of suppression of, 107
- Visually directed movements, 245
- Von Frey's hairs, 47, 48
- Walking in tabes, 258, 260
- Wassermann reaction in tabes, 152
- Wilson, anterior horn cells in tabes, 13
- Wittmarck's experiments, 111-112
- Wolf, cure of ataxia, 274

LANE MEDICAL LIBRARY

To avoid fine, this book should be returned on
or before the date last stamped below.

MAR 14 1949

DEC 10 1918

JAN 16 1918

JUN 10 1918

SEP 2 1921

MAY 10 1922

MAR 23 '23

6/2/52

OCT 24 '23

DEC 10 '23

DEC -5 '24

AUG 29 '28

JAN 17 1934

NOV -6 1946

MAR 1947

L409	Maloney, W.J.M.A.,
M25	Locomotor ataxia
1918	(Tabes dorsalis)

45327

NAME	DATE LUE
Sam Anderson	MAR 8 1944
Betty Steele P.T.	3/10/44
J. Dennis P.T.	6/2/52

NAME
San Antonio

DATE DUE

Betty Steele

P.T.

3/16/49

A. Bernis P.

97

6/2/57

